Diseases of the Heart

Diseases of the Heart:

Their Diagnosis and Treatment.

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CONTENTS.

PREFACE.

This little book was never intended to aspire to the dignity of a treatise on diseases of the heart. The primary object was to make it useful to the practical physician in the diagnosis of cardiac diseases. The cardiac diagnostician is often like the veterinarian, for his diagnosis is based essentially on objective signs. He must depend largely on the Baconian or inductive method of ratiocination, in contradistinction to the deductive method.

The former analytic method of diagnosis is a conclusion drawn from concrete facts. Mistakes in diagnosis may be attributed to the following causes: 1. Incomplete or careless examination. 2. Misinterpretation of symptoms, due to errors in judgment. 3. Ignorance of the methods of examination. 4. Prejudiced preconception. 5. Incompleteness of medical diagnosis. 6. Placing too much reliance on the results of treatment. 7. Incomplete history of the case, and the incomplete development of symptoms. 8. Simulation or dissimulation on the part of the patient.

1. Errors in diagnosis are not so much due to ignorance as carelessness. Sir William Savory tritely remarks, "Consciousness of one's ignorance may do much to avert the errors of carelessness, and he who has confidence in his own judgment should of all men be most careful in inquiry."

Unfortunately, we of to-day treat the disease, but not the patient. "And I said of medicine, that this is an art which considers the constitution of the patient, and has principles of reason and action in each case." It is but a few years ago, that a physician punctured a pregnant uterus with a trocar, believing that he was dealing with a case of ascites. We recall the grave error occurring in the practice of a famous English surgeon who mistook a swelling in the neck for an abscess, who, with more precipitation than reflection, plunged his lance into the tumor and death from hemorrhage resulted.

2. Under the caption of misinterpretation of symptoms due to errors in judgment, mistakes may arise from (a) placing too much reliance on the subjective symptomatology; (b) giving undue prominence to one symptom to the exclusion of others; (c) grouping symptoms which are the effect of disease, and not the disease itself. When the pathologist makes an autopsy he records many of the pathological conditions found, as anatomic diagnoses. The clinician should be similarly guided. It would appear at times as if, in our struggle to establish a diagnosis, it would be better to make none at all, rather than group symptoms under such equivocal expressions as pseudoangina, arrythmia, cardiac palpitation, etc. Such expres-

sions mean practically nothing in etiologic diagnosis.

3. Ignorance of the methods of examination is responsible for many unfortunate mistakes. The rejected applicants of insurance companies furnish a large contingent. Nephritis is diagnosed because albumin is present in the urine, diabetes, because sugar is found, and heart disease because murmurs are heard. An unprincipled physician could reap a harvest, by putting in condition for re-examination many rejected applicants, diseased or otherwise, for life insurance.

4. Prejudiced preconception arises from two causes: (a) Placing too much reliance on the history of the patient; (b) being misled by first appearances. Like the critic who never read a book before he received it because he might be prejudiced, so it should be with the physician—he should not learn the history of his patient before he examines him. Diseases present such various pictures, that with our mental astigmatism, we can see anything we want. The personal history of the patient should only be used in confirming the objective examination.

5. When a disease runs a typic course diagnosis is, as a rule, easy; but when the affection is atypic, one is frequently led into error. The physician is too often inclined to misinterpret the limitations of his art, mistaking the latter for his own delin-

quencies. Myocarditis is more often an anatomic than a clinic diagnosis. Differentiation between cardiac dilatation and pericardial effusion is exceedingly difficult at times and to puncture the dilated heart with the idea that the latter condition is present is a gross error. Treatment should never be attempted before a diagnosis is made. Better no treatment than meddlesome therapy. *Qui bene dignoseit, bene curat.* It is related of Frerichs, that after examining a patient, he was in doubt about the diagnosis. The patient insisting about knowing the nature of his trouble, Frerichs comforted him with the assurance that the diagnosis would be determined at the autopsy.

6. We are frequently led into error by mistaking recovery for cure, thereby ignoring the *vis medicatrix naturae.* I have seen many patients with organic cardiac murmurs, the latter becoming less intense after the administration of chalybeates. Under the circumstances, one would be inclined to regard the murmurs as anemic. Upon more mature consideration, this view would be dispelled. Impoverishment of the blood attends nearly all organic cardiac affections and only succeeds in intensifying the murmurs, hence iron only removes the factor in intensification.

7. Diagnosis must be held in abeyance in many cases owing to undeveloped symptoms and incomplete history of the case. Problematic diagnoses

are elusive, and a diagnosis altered to correspond with each stage of the patient's illness is no diagnosis at all.

8. Disease is expressed in a manner peculiarly its own. The interpretation of the signs constitutes diagnosis. The translation may be correct, partially correct, or wrong. In all three instances the result, as far as the patient is concerned, will, as a rule, be the same, provided no treatment is instituted. To treat a disease, other than by expectant methods, where the diagnosis is wrong, is adding insult to injury.

S. W. cor. Van Ness Avenue and California St.
December, 1900. San Francisco.

CHAPTER I.

INTRODUCTION.

The heart with its valvular apparatus acts like a pump with a suction and pressure valve. During diastole, it sucks the blood from the veins, and during systole drives it into the arteries. Therefore during diastole the pressure in the veins sinks and rises in the arterial system during diastole. This difference in pressure causes the blood to circulate.

COMPENSATION.

All heart affections, whether of the valves, muscle or pericardium, result in circulatory disturbances and are characterized by diminished pressure in the arteries and increased pressure in the veins, with retardation of the blood current in the capillaries. When the heart by increase of power and volume opposes the local and general disturbances, the lesion is said to be *compensated,* and a well compensated valvular lesion may be unattended by subjective symptoms.

Compensation fails when the heart muscle (myocardium), in consequence of nutritive disturbances, degenerates. A valvular heart trouble, especially in children, retards development and

nutrition, leading to *cardiac cachexia.* The notable tissue changes are thickening of the nose and lips and clubbing of the finger ends.

Overloading of the veins leads to the accumulation of fluid in the tissues; beginning first in the feet, it gradually invades the rest of the body. Fluid also accumulates in the serous cavities (pleura, pericardium, brain ventricles). As a rule, the peritoneum is the first serous cavity invaded (ascites). The chief cause of cardiac dropsy is disease of the mitral valve, and especially *mitral stenosis.*

Cyanosis of the skin is an early sign and appears as soon as the pulmonic circulation is disturbed, therefore cyanosis is more evident in mitral than in aortic lesions. The cutaneous veins are filled with blood and may become varicosed. *Jaundice,* due to catarrh of the bile passages, is not uncommon. *Cutaneous hemorrhages* from rupture of the capillaries or caused by emboli may develop.

The temperature of the body may be normal or lowered, owing to the retarded circulation. Intercurrent elevations of temperature may be caused by emboli in the viscera or lung infarcts.

PULSE.

The pulse has a specific character in nearly every valvular lesion. Disturbance of compensation gives a frequent, irregular, soft and feeble pulse. An

intermittent pulse is caused by feeble heart contractions which are not strong enough to drive the blood to the radial artery. In such instances, if the heart is auscultated synchronously with palpation of the pulse, there are more heart tones than pulse beats.

Palpitation of the heart, a frequent symptom, may be subjective, objective, or both. *Pain* in the precordia radiating to the left arm, neck or umbilicus, gives rise to symptoms not unlike angina pectoris. This *precordial pain* is especially frequent in aortic incompetency and has been attributed to irritation of the cardiac plexus by the dilated aorta.

BLOOD-VESSELS.

Emboli and thromboses occur. Emboli from the right ventricle pass into the pulmonary arteries and cause hemorrhagic infarctions. Emboli originating from the left ventricle go to the extremities, skin, retina or the viscera. Embolism of the spleen is manifested by a sudden chill, fever, perspiration, pain in the splenic region and enlargement of that viscus.

To the foregoing symptoms, hematuria is added when the embolus attains the kidney. An embolus of the brain reaches that organ usually through the left carotid artery.

LUNGS.

Dyspnea, especially on exertion, is frequent. The dyspnea of heart disease is out of all propor-

tion to the physical changes in the lungs. Difficult breathing is usually caused by pressure of the enlarged heart on the lungs, disturbed pulmonic circulation, hydrothorax, ascites or bronchial catarrh.

Hemoptysis occurs frequently in mitral disease. Hemorrhage may be due to congestion, rupture of vessels or hemorrhagic infarcts. Hemoptysis is most frequently the result of infarcts, and the latter are frequent in aortic disease. Lung infarcts lead to a brownish red sputum not unlike that of pneumonia. Stress has been laid on the fact that in hemoptysis of cardiac origin, the blood is clotty and blackish blue in color.

Edema of the lungs is a frequent cause of death. It gives rise to diffuse crepitant rales and serous expectoration. Valvular heart troubles predispose to inflammatory lung affections. Glottis edema may complicate heart lesions. Epistaxis is not infrequent.

GASTRECTATIC DYSPNEA.

A frequent cause of dyspnea in heart disease is *acute dilatation of the stomach.* After meals patients complain of difficult breathing and distress in the precordia, and death has not unfrequently followed an indigestible meal. I have called this condition *gastrectatic dyspnea,* because it is always associated with a dilated stomach. In some instances dyspnea is associated with symp-

toms of *angina pectoris.* Many patients make no mention of dyspeptic symptoms. They complain of pressure or weight in the sternal or precordial region, and often add that eructation will relieve the pressure. This symptom, as I have assured myself after examination of a number of cases, is dislocation of the heart upwards by an acute or chronically dilated stomach. Some years ago I reported a case of gastroptosis and merycismus, with voluntary dislocation of the stomach and kidneys.* This phenomenal case taught me one fact in particular, how easily the heart could be displaced by dilatation of the stomach. The individual in question could, by buccal insufflation of the stomach, cause his heart to disappear behind the lungs, so that percussion of the precordial region yielded no dullness on percussion. This case directed my attention to a correct investigation of all individuals presenting themselves for the treatment of slight dyspeptic symptoms in whom sternal pressure was the chief subjective symptom.

In all such cases the diminished area of cardiac dullness bears a distinct relation to the severity of the pressure symptoms. The removal of ingesta and gases from the stomach restores the heart to its normal position and feeble heart tones become strong.

*Medical News, April 13, 1895.

Not infrequently true asthmatic attacks, *asthma dyspepticum,* were present. The patient is unable to get rid of the gases owing to a spasm of the

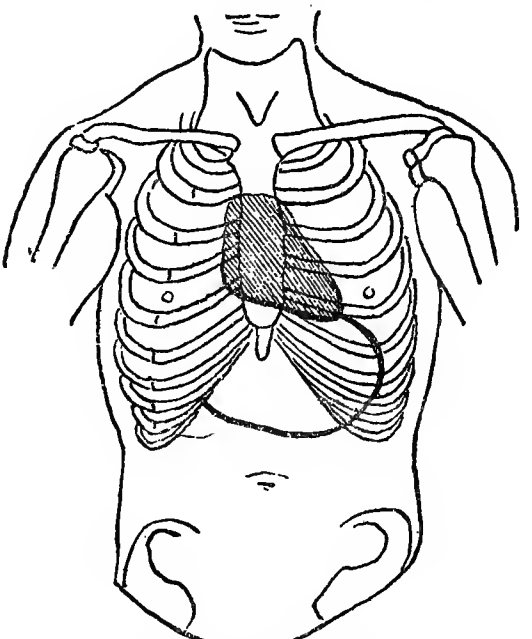


Fig. 1—Skiascopic picture of the outline of the heart and stomach before swallowing the seidlitz powder.

sphincters of the stomach; the distended stomach pushes the diaphragm upward, dislocating the heart, and induces typical attacks of asthma.

To quickly detect a dilated stomach encroaching on the chest organs, the following percussion method will be found practical. The circular tympanitic stomach-lung region formed by the stomach beneath the lower lobe of the left lung gradually disappears behind the axillary line if the stomach is normal, but if dilated, the tympanitic sound may be traced to the vertebral column. Sometimes in dyspeptic asthma relief is quickly obtained by introduction of the stomach tube and allowing the gases to escape. The following illustrations describe more fully than words the influence of a dilated stomach on the position of the heart. They are rough reproductions from the fluoroscopic picture with the use of the X-rays. In the average examination of the chest by the X-rays, the portion of the stomach which is in direct contact with the chest wall is obscured by the shadow cast by the spleen, but in this patient no spleen shadow being present and the contour of the stomach being clearly defined, opportunity was afforded to test the influence of a stomach distended by a seidlitz powder on the position of the heart.* Every phase of the stomach distention was followed in the fluoroscopic picture.

*Later a similar case came under my observation. See "Note on a Case of Nervous Eructations Studied by Skiagrams," Philadelphia Med. Journal, Aug. 12, 1899.

CARDIAC ASTHMA.

Cardiac Asthma closely simulates bronchial asthma, but the former is associated with some anomaly of the heart or arterial system. If such

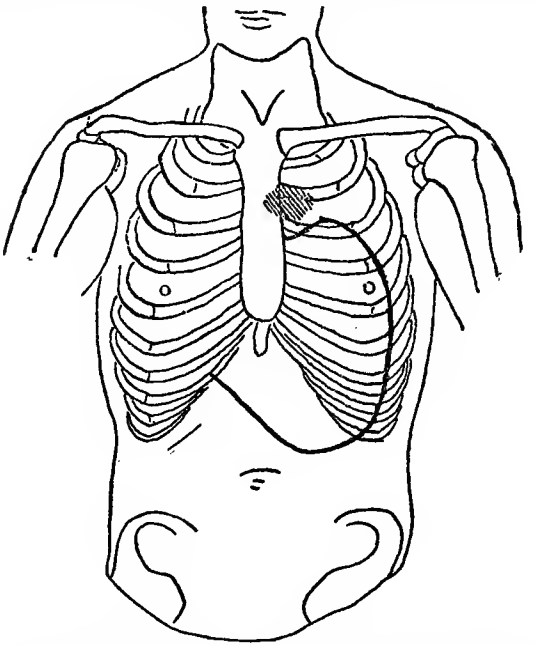


Fig. 2—Shows the same organs after distention of the stomach by gas.

anomalies exist, asthmatic paroxysms may result, whenever the pressure in the capillaries of the lungs rises. Such rise in pressure may follow an

increased or diminished blood pressure in the aorta. In either instance, the capillaries of the lung alveoli become surcharged with blood, which in turn make the alveolar walls rigid and incapable of distension, thus diminishing the respiratory area. The following table may assist in differential diagnosis:

CARDIAC ASTHMA.	BRONCHIAL ASTHMA.
Signs of cardiac disease (valvular lesion, arterio sclerosis, fatty heart).	Usually absent.
Dyspnea is equally inspiratory and expiratory.	Dyspnea is expiratory.
Pulse in the early stage of paroxysm may be strong, but it soon becomes soft and small.	The pulse is usually one of increased tension throughout the paroxysm.
Percussion shows an extension of the borders, of the lungs and obliteration of the area of superficial cardiac dullness.	The extension of the lung borders is more pronounced than in cardiac asthma.
Auscultation shows an abscence of râles unless complicated by edema of the lungs.	Sonorous and sibilant râles are always heard, louder during expiration than inspiration.

DIGESTIVE APPARATUS.

Venous stagnation conduces to chronic catarrh of the gastro-intestinal mucous membrane, resulting in dyspepsia, constipation, diarrhea and

hemorrhoids. Gastralgia occurring in cardiac lesions may mislead the physician if the diagnosis is of a stomach trouble.

LIVER AND SPLEEN.

The liver participates early in the circulatory disturbances. Owing to the venous engorgement of the inferior cava, the hepatic veins cannot unload, and the liver in consequence swells and may be felt below the border of the ribs as a hard and painless mass. Later in the disease, owing to atrophy of the liver cells, the organ may become reduced in size. Not infrequently the enlarged liver may pulsate owing to *transmitted pulsations* from the aorta. It is well to remember that the knee-elbow position will usually cause the disappearance of transmitted pulsations. Stagnation of blood in the portal circulation leads to venous engorgement of the spleen, stomach and intestines, with enlargement of the first mentioned viscus.

KIDNEYS.

From the quantity and constituents of the urine the severity of the compensation failure may be gauged. The lower the blood pressure in the aorta and the higher the blood pressure in the venae cavæ, the more the urine partakes of the characteristics of passive congestion of the kidneys. The urine is reduced, of high specific gravity, contains albumin, casts, and often blood corpuscles. Uric

acid is increased and is deposited as a brick dust sediment.

NERVOUS SYSTEM.

Aortic lesions, particularly owing to brain anemia, are often complicated by syncopal attacks. Brain hyperemia complicating heart lesions is characterized by attacks of fainting, fullness in the head, ringing in the ears, etc. *Nitrite of amyl* inhalations are of signal advantage in diagnosis. This drug will ameliorate symptoms of brain anemia and intensify those of hyperemic origin. An embolus in the left arteria fossae sylvii will cause hemiplegia on the right side, associated with aphasia. *Temporary aphasia* may occur without an embolus and must often be attributed to mere circulatory disturbances. Mental diseases are not frequent in heart lesions. In some cases a real intellectual disturbance exists. Observations are recorded of maniacal delirium in patients with mitral lesions. Such cerebral troubles may be remedied by treatment directed exclusively to the heart.

RELATION OF DISEASES OF THE HEART TO OTHER DISEASES.

An individual with a heart lesion assumes a grave risk when attacked by other diseases. This is notably the case in *febrile affections.* In fever, the organs show cloudy swelling; a like change occurs in the muscles, and the heart manifests the

granular alteration of its fibres to the highest degree. These tissue changes arise from contact with the poisons circulating in the blood and from the accompanying rise of temperature associated with disturbances of nutrition. A febrile affection therefore may seriously implicate the functions of the heart in valvular lesions.

Intercurrent diseases of the lungs tax the functions of the right heart to the utmost.

Pregnancy always causes hypertrophy of the heart, but this recedes in the healthy woman after delivery. Cardiopathic patients are predisposed to acute exacerbations of endocarditis, and a large number are always in danger of miscarriage. Durosier noted that out of forty children born of cardiopathic mothers, thirty-seven died before attaining the age of six years. The most unfavorable lesion to the mother from the point of prognosis is mitral insufficiency, the mildest, aortic insufficiency. The most serious complications, and the greatest danger of death for the mother, appear about the seventh and a half, or the eighth month. Cardiopathic mothers should not nurse their infants because lactation augments heart hypertrophy.

Endocarditis is regarded by some as the cause of *chorea;* particles of fibrin are supposed to pass from the valves as emboli to the cerebral vessels. At any rate, endocarditis is very common as a

complication, although many of the heart murmurs in chorea may be caused by anemia or the rapidly acting heart.

The belief was at one time current that an individual with heart disease was in no danger of contracting phthisis. As a rule (*pulmonary stenosis* the exception), pulmonary tuberculosis rarely develops in an individual with a valvular heart lesion. In 277 autopsies on individuals who during life suffered from valvular trouble, Frommalt found phthisical lung changes in 8 per cent of the cases. These statistics show the infrequency of phthisis complicating valvular lesions, since Biggs reports that more than 60 per cent of his autopsies showed lesions of pulmonary tuberculosis.

ETIOLOGY OF DISEASES OF THE HEART.

Endocarditis is the usual cause of valvular heart lesions. That part of the endocardium performing the most work is the first to become involved and suffer most. This explains the rarity of endocarditis on the right side in adults and the infrequency of congenital lesions on the left side of the heart. The process usually implicates the valvular endocardium and is therefore known as *valvular endocarditis.* In adult life, about one-half the cases of endocarditis occur on the mitral valves; of the remaining 50 per cent, about 94 per cent occur on the aortic valves; the remaining cases are divided between the valves of the right side, the

tricuspid valve being in the ascendency. It is customary to speak of the following forms of endocarditis:

(a) Acute { simple. / malignant.

(b) Chronic or indurative.

(a) The acute *simple endocarditis* is caused by acute articular rheumatism in 20 per cent of the cases. Among the other causes are: the infectious diseases of children, tonsillitis (by many regarded as the avenue of rheumatic infection), pneumonia, and diseases associated with blood intoxications, like diabetes, gout, cancer, and nephritis, especially the interstitial form. Various organisms, like strepto- and staphylococci, gonococcus, and even the bacillus tuberculosis, have been found in and on the affected valves, but their casual relationship has not been demonstrated.

The *malignant form* is of microbic origin and is secondary to some infectious disease. The majority of cases develop during an attack of croupous pneumonia. The other diseases associated with the infectious process are: pyemia, septicemia, puerperal fever, gonorrhea, erysipelas, puerperal fever, diphtheria and rheumatism.

(b) Chronic endocarditis results from the acute forms and from syphilis, alcoholism, gout and excessive work for any one valve.

RESULTS OF ENDOCARDITIS.

When restitution of the valve does not take place (rare), one of two conditions of clinical importance occurs, narrowing, obstruction or stenosis, or insufficiency or incompetency of the valves. In either instance, murmurs are heard resulting from obstruction to the onward flow of the blood or from leakage backwards through a closed but incompetent valve. The former are known as *obstructive,* the latter as *regurgitant* murmurs.

RESULTS TO THE HEART.

The inevitable consequence to the heart in a valvular lesion is increased work, leading to *hypertrophy* or *dilatation.*

Hypertrophy is muscular thickening of the walls of one or more cavities of the heart, and rarely occurs without some dilatation of the cavities. Increased work of the heart, when nutrition is plentiful, is followed by hypertrophy. Overwork, beyond the nutrition and muscular power of the heart, results in dilatation. Hypertrophy is a favorable compensatory condition in cardiac lesions; it is the response of the cardiac muscle to an increased demand for power. It can only develop when the health of the organism is maintained at the proper standard, and when this fails the compensation attempted by nature must fail, and then hypertrophy passes into dilatation.

Heart strain is a prolific etiologic factor in dis-

eases of this organ and of the aorta. The initial effect of *prolonged exertion* is dilatation of the right side of the heart. The effect of sudden strain is on the aortic area. Peacock found, in 17 cases of rupture of the heart valves after sudden strain, that the aortic valves were implicated ten times, mitral valves four times, and the tricuspid valves three times. Schott* has demonstrated in a series of skiagraphs that dilatation of the heart after wrestling can be demonstrated by the Roentgen rays.

In recent years, heart disease, resulting from overstrain after bicycling, has been frequently observed. I have examined a few individuals with the X-rays who have done "century runs," and have demonstrated dilatation of the right heart following such foolhardy attempts. I have personal knowledge of five individuals who have become heart cripples from excessive bicycling.

The size of the heart chambers varies in health. In severe exertion the chambers dilate, especially those of the right side, to accommodate themselves to the increased quantity of blood; this compensation on the part of the heart is "the getting of wind," as it is called in training. When an individual in poor condition subjects himself to heart strain he suffers from rapid and feeble pulse, cardiac dyspnea and precordial pain, and for months

*Medical Record, March 26, 1898.

after he may be unfitted for severe exertion or became permanently crippled. Systematic and judicious muscular exercise develops heart hypertrophy, a propitious condition when great endurance is demanded. Injudicious exercise weakens the heart.

Relative valvular insufficiency (i. e., normal valves which are no longer capable of completely closing the orifices of the heart), especially of the tricuspid valves, frequently follows heart strain. In men the aortic valves are more frequently implicated than in women. This is owing, no doubt, to the fact that bodily exertion predisposes to arterial disease. Among the laboring classes valvular lesions are most frequent.

FREQUENCY OF INDIVIDUAL VALVULAR LESIONS.

In extra-uterine life the most frequent valvular lesion is mitral insufficiency, then follows mitral stenosis, combined with mitral insufficiency, then aortic insufficiency, then aortic stenosis, and finally aortic stenosis combined with aortic insufficiency. Combined lesions are not infrequent. Mitral and aortic lesions may coexist and less often mitral and tricuspid lesions. In children, the most common combination is aortic and mitral insufficiency.

PROGNOSIS OF DISEASES OF THE HEART.

The prognosis in valvular lesions is unfavorable. Cure may be spontaneous, but is never attained by

medication. Aortic are more favorable than other lesions, owing to the ability of the voluminous left ventricle to compensate the defect. Pulmonary lesions are especially unfavorable, owing to the frequency of phthisis complicating such lesions. Combined lesions of different valves are more unfavorable than lesions of individual valves, owing to the increased work thrown on the heart. The social position of the patient influences the prognosis. Occupation which demands little muscular effort and permits a sedentary life favors longevity. The stronger the constitution the greater the likelihood of the heart being able to meet the increased demands made on its power. Valvular lesions acquired in childhood soon result in compensatory disturbances.

Mechanical troubles of circulation when the heart muscle is inadequate to perform its task furnish an unfavorable prognosis and lead to a lingering illness, death resulting eventually from paralysis of the heart, blocking of one of the branches of the coronary arteries, lung edema or debility. In other instances death is sudden from heart rupture or cerebral complications. So long as an efficient compensation is maintained in valvular disease, even the most serious valve lesion is unattended by inconvenience to the patient. Sir Andrew Clark summarized the following conditions which justified a favorable prognosis: Good

general health; just habits of living; no exceptional liability to rheumatic or catarrhal affections; origin of the valvular lesion independently of degeneration; existence of the valvular lesion without change for over three years; sound ventricles, of moderate frequency and general regularity of action; sound arteries, with a normal amount of blood and tension in the smaller vessels; free course of blood through the cervical veins; and lastly, freedom from pulmonary hepatic and renal congestion.

CHAPTER II.

THE DIAGNOSIS OF DISEASES OF THE HEART.

SIGNIFICANCE OF MURMURS.

No fallacy in medicine has been more carefully nourished than the belief that a cardiac murmur is always indicative of heart disease. Some of the most serious heart affections are unaccompanied by murmurs. "The idea that a murmur in itself and by itself is a serious thing dies hard" (Shattuck). Sir Andrew Clark gave utterance to the truism "that a murmur in itself is of little or no moment in determining the prognosis of any given case. Osler voices the opinion of the skilled cardiac diagnostician as follows: "Practitioners who are not adepts in auscultation and feel unable to estimate the value of the various heart murmurs should remember that the best judgment of the conditions may be gathered from inspection and palpation. With an apex beat in the normal situation and regular in rhythm, the auscultatory phenomena may be practically disregarded."

THE APEX BEAT.

We must always remember that disease of the heart valves of any consequence to the patient,

always leads to functional and structural heart changes and unless the latter can be demonstrated, the diagnosis of valvular disease should be held in abeyance. Fowler is responsible for the epigram: "That the position of the cardiac apex is the key to the diagnosis of nearly all affections of the chest and heart."

The normal location of the apex beat excludes dilatation, hypertrophy, pericardial effusion and heart dislocation.

CARDIAC MURMURS.

Adventitious sounds originating in the pericardium heart and blood vessels are known as *murmurs*. The auscultation of a murmur suggests many problems in diagnosis. Having determined the presence of a murmur the first problem to unravel is its origin. The most frequent murmurs are endocardial in origin and they are divided into organic (if caused by anatomic changes of the heart or blood vessels) and inorganic or functional murmurs (caused by changes in the quality of the blood. An *organic murmur* may be obstructive or regurgitant. Two problems await solution: First, the seat of the murmur; second, the nature of the murmur.

The seat of the murmur is determined by noting its position of maximum intensity and the direction of its transmission. These facts apprise us of the valve orifice affected.

THE ORIFICE AFFECTED.

The position of maximum intensity of a murmur usually occurs at the point where the normal valve sound is best heard in health. We must not forget that the heart orifices are closely situated and therefore murmurs are created within a limited area; if it were not for the fact that murmurs have directions of selective propagation it would be impossible to determine at which valve orifice the murmur was generated.

DIRECTION OF TRANSMISSION, NATURE AND TIME.

In general, systolic murmurs of aortic origin are transmitted upwards from the base. Systolic murmurs of mitral origin are transmitted toward the axilla. The transmission of a murmur is in the direction of the currents which produce them.

Our next duty is to determine the nature of the murmur, which is ascertained by noting the time of the murmur and the direction of its propagation. Organic endocardial murmurs may be obstructive when there is obstruction to the onward flow of blood, the nature of the lesion being a stenosis and regurgitant murmurs when there is leakage backwards through a closed but incompetent valve, the nature of the lesion being an insufficiency.

Organic heart murmurs have a definite relation to the cardiac cycle and we distinguish *systolic, diastolic* and *presystolic murmurs.*

SYSTOLIC MURMURS.

The systolic murmurs arise from aortic obstruction, and mitral and tricuspid regurgitation. Systolic murmurs are synchronous with the carotid pulse, therefore in a rapidly acting heart, the time of the murmur may be determined by palpation of the carotid pulse during auscultation. The radial pulse should not be selected because it is felt too long a time after systole.

The diastolic murmurs are aortic regurgitation, and mitral obstruction. The so-called presystolic murmur is associated with mitral stenosis: it occurs at the end of systole, or, in case it is present at the beginning of diastole, it becomes stronger toward the end.

CHARACTER OF MURMURS.

Regurgitant murmurs as a rule are soft and blowing. The murmur of aortic regurgitation is characterized by length and softness, while the murmur of mitral regurgitation is louder, but not so long. Murmurs that are rough and high in pitch are usually generated by valves which are thickened and rigid, a common condition in chronic endocarditis. Murmurs soft and low in pitch are associated with soft exudations on the valves and are heard in endocarditis of rheumatic origin. The murmur of mitral obstruction is the only murmur which has a specific character. It

is a prolonged murmur of a churning or grinding character as if fluid were being forced with great effort through a narrow channel.

Murmurs may sometimes be felt in the heart region. The sensation is similar to that perceived upon stroking the back of a purring cat; for this reason, they are called *purring tremors.* Like murmurs, they may be presystolic, systolic, or diastolic in time. They are nearly always indicative of a valvular lesion.

SECONDARY EFFECT OF VALVE LESIONS.

Having ascertained the endocardial character of the murmur and the seat of the lesion our next endeavor is to confirm our diagnosis by determining the all important fact, viz.: the secondary effect of the lesion on the heart. Without this corroboration the detection of a murmur is without diagnostic or prognostic importance.

Aortic Obstruction.—Owing to the obstruction of blood from the left ventricle, the latter must work with increased force, therefore it hypertrophies. Less blood on account of the stenosis is thrown into the arterial system, hence the pulse is small and of high tension owing to the hypertrophied left ventricle. *Aortic Regurgitation.*—The blood flowing back into the left ventricle during diastole, causes this chamber of the heart to enlarge (dilatation), but compensation occurring, the dilatation is overcome by hypertrophy of the

ventricle. The pulse of aortic regurgitation is pathognomonic. It is called the *Corrigan* or "water hammer pulse." The impression received by the finger on the radial artery is one of recedence

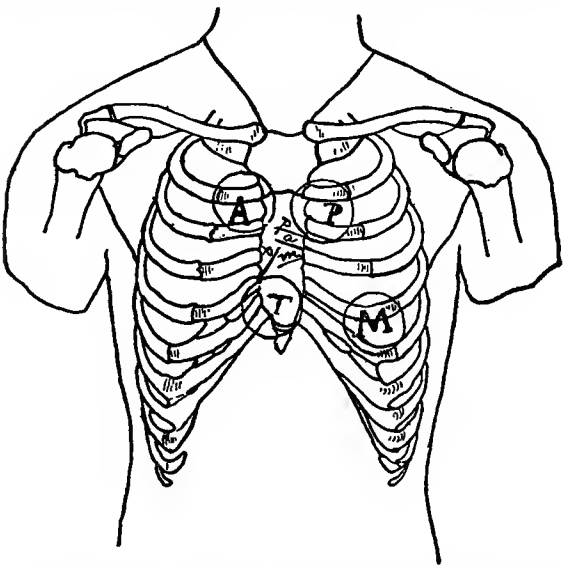


Fig. 3—Auscultatory areas of the valves and points of maximum intensity of the murmurs: M, mitral valve; T, tricuspid; P, pulmonary; A, aortic. Anatomic position of the cardiac valves: t, tricuspid; m, mitral; a, aortic; p, pulmonary.

of the pulse wave as soon as it strikes the finger. The phenomenon is accentuated if the arm is raised.

Mitral Regurgitation.—In this lesion the brunt of the work is thrown on the right ventricle, which dilates and hypertrophies. The increased tension of the pulmonary artery is evidenced by accentuation of the second pulmonic tone. The arterial system receives less blood leading to insufficient nourishment of the heart through the coronary arteries, hence degeneration of the organ must ensue.

In *Mitral Obstruction* it is the left auricle which primarily hypertrophies to overcome the narrowed mitral orifice. Later, the right ventricle hypertrophies.

ACCIDENTAL HEART MURMURS.

There are a number of accidental heart murmurs, functional in their nature, which admit of no definite classification. As a rule, they are unattended by any palpable changes in the heart or pulse. They are almost invariably *systolic* in time. In my experience, they are frequent before operations and in gastric disturbances. There are many individuals, chiefly women in whom functional murmurs appear just before an expected operation and disappear with equal readiness a few days after the operation. They might correctly be called *"murmurs of apprehension."*

The other class of murmurs associated with stomach disturbances, which for purposes of con-

venience I will designate as *"murmurs of gastric origin,"* I have encountered frequently. They usually coexist with digestive disturbances and are sometimes of great intensity. Such individuals complain of precardial pain and pressure and the disappearance of the latter symptoms mark the evanescence of the heart murmurs. The murmurs are in no wise associated with the pressure of a dilated stomach on the heart as would be primarily surmised, for I have never been able in such individuals after disappearance of the murmurs to recreate them by artificial insufflation of the stomach. Other causes must exist and the most likely cause is reflex irritation of the cardiac nerves superinduced by the toxic products of gastric indigestion. While stress has been laid on the fact that functional murmurs are in the great majority of instances systolic in time, we must not forget that they may also be diastolic. In my experience I have encountered such murmurs in anemia, with their maximum intensity over the auscultatory situation of the aortic orifice and they may be traced to the jugular veins in the neck, their undoubted point of origin. Care must be exercised in distinguishing such murmurs from those occurring in aortic incompetency, an error which is hardly possible, if all the facts in this chapter are carefully considered. The foregoing facts prompt us to hold in reserve the diagnosis, "or-

ganic heart murmur," without repeated examinations of the heart, for it is evident that, if at one examination, we note, let us say, a systolic murmur at the mitral area and at a subsequent examination a systolic tone, as a rule there can exist no organic disease of the valve.

ANALECTIC REVIEW OF CARDIAC VALVULAR MURMURS.

1. The character or intensity of a murmur is no index to the gravity of the lesion producing it. The loudest murmur may be produced by the smallest lesion and vice versa.

2. The loudness of a murmur is largely dependent on the activity of the heart. Loud murmurs may become weak, and this change is an ominous sign indicating heart weakness. For the same reason they may disappear in febrile diseases and in the dying state. Faint may often be converted into loud murmurs after increasing cardiac activity by exercise and cardio-tonic medication. Complete compensation may often cause the temporary disappearance of a murmur.

3. In some individuals murmurs are louder in the recumbent than in the erect posture, especially murmurs of tricuspid and mitral origin. Murmurs should be auscultated with the patient in different postures.

4. Murmurs are less loud in inspiration than expiration.

5. Strong pressure on the chest, especially in children, may cause the disappearance of murmurs, the pressure inhibiting cardiac action.

6. When the heart is rapid or irregular in action, it is difficult to determine the time of a murmur. Remember that systolic murmurs are synchronous with the carotid pulse. Also regulate the action of the heart with digitalis.

7. Systolic are usually louder though less prolonged than diastolic murmurs.

8. When murmurs are faint, have the patient suspend respiration during auscultation.

9. Murmurs are most intense at their point of origin and they are propagated in the direction of the blood current by which they are developed.

10. Murmurs of extra-uterine origin are oftener found to proceed from the valves of the left heart, and in adults, murmurs at the tricuspid and pulmonary areas are rare.

11. In rare cases the murmur may be heard at a distance without laying the ear over the chest and they may be perceived by the patient. Only those arising at the aortic opening have this peculiarity.

12. When two murmurs co-exist at systole or diastole they may be transmitted or be due to disease at different orifices. Thus two murmurs oc-

TABULAR VIEW OF ORGANIC AND INORGANIC HEART MURMURS.

NATURE OF MURMURS	CAUSE	WHERE HEARD	CHARACTER	DIAGNOSIS
Endocardial Organic Murmurs.	Disease either of or close to the valve where they occur.	At a point where the valve sound is best heard in health.	Obstructive murmurs are apt to be rough and regurgitant, soft and blowing.	1. Time of occurrence. 2. Point of maximum intensity. 3. Direction of transmission. 4. Character.
Pericardial Murmurs.	Rubbing together of the roughened surfaces of the pericardium.	Appears first near base of the heart, but, when pericarditis is general, best heard near the left nipple.	Sounds of a peculiar, rubbing, grating quality or of a creaking quality, like new leather.	Variability in position and quality of sounds which are not synonymous with the heart sounds and their superficial character.
Murmurs of incompetency with healthy heart valves.	The ventricular cavity enlarges so that the valve does not coaptate to close the widened orifice.	At a point corresponding to the valve sounds.	Soft and low in pitch and not widely transmitted.	The heart is dilated. When dilatation is overcome the murmur disappears.
Anemic Murmurs.	Many ascribe murmurs to slight dilation of pulmonary artery and to less vicious state of the blood.	Systolic murmur at the base or apex, more often in the former situation. Loudest in the second left interspace.	Soft in character, and loudness increased by violent cardiac action	Temporary. Disappear after cure of the anemia.
Cardio - Respiratory Murmurs.	A rapid and vigorous heart forcing the air out of the lung over the left ventricle.	To the left of the apex. Often heard in the second left interspace about 2 in. from sternum.	Resembles ordinary expiratory murmur; is of greatest intensity when lung is full of air.	Frequently disappear when the breath is held in deep expiration and when the patient is lying down.
Cardio - Muscular Murmurs.	Excited action of the heart.	Loudest at the fourth left interspace close to the sternum.	Rough or whizzing. Increased by exertion and excitement.	Longest at end of expiration. Disappears when inspiration ends or patient lies down.

curring at systole may be due to mitral insufficiency and aortic stenosis or if occurring during diastole, to mitral stenosis and aortic insufficiency. Differentiation is possible in two ways: First, by the character of the murmur. If one is blowing and the other rough, two distinct murmurs exist. If both are similar in character, then there is only one, which is transmitted from its point of origin at one opening to the second opening.

Second: Auscultate from the point where one murmur is heard to where the other exists, as from the apex to the aorta. If the murmur is everywhere distinct but it becomes gradually louder toward one point, then it arises at this point and is transmitted to other points. If, on the contrary it is no longer heard at some point between the apex and the aorta, and is again audible at the aorta, then there are two murmurs.

13. Never diagnose a valvular lesion without taking into consideration the effects of such a lesion on the heart and blood vessels and demonstrating them.

PERICARDIAL MURMURS.

These are friction sounds produced by the rubbing of one surface of the pericardium upon the other when roughened by a fibrinous exudate which occurs in the plastic variety of pericarditis. The following characteristics will aid in distin-

TABULAR REVIEW OF CARDIAC VALVULAR MURMURS.*

TIME OF MURMUR	USUAL SEAT OF MURMUR	TRANSMISSION OF MURMUR.	CAUSE OF MURMUR.
Aortic Orifice—Systolic Murmurs. (Aortic obstruction.)	Second right intercostal space close to the border of the sternum.	Over the carotid arteries. The most widely diffused of all cardiac murmurs.	Obstruction to outward flow of blood through aortic orifice.
Mitral Orifice—Systolic Murmurs. (Mitral regurgitation.)	At the apex.	Axilla and angle of scapula. Sometimes heard loudest along left border of sternum.	Regurgitation of blood through mitral orifice into left auricle.
Pulmonary Orifice—Systolic Murmur. (Pulmouary obstruction,)	Second left intercostal space close to sternal border.	Murmur is not transmitted to the vessels of the neck.	Obstruction to outward flow of blood through pulmonary orifice.
Tricuspid Orifice—Systolic Murmur. (Tricuspid regurgitation.)	Median line of sternum on a level with fifth intercostal cartilage.	Limited in transmission to the limits of percussion dulness.	Regurgitation of blood through tricuspid orifice into right auricle
Aortic Orifice—Diastolic Murmur. (Aortic regurgitation.)	Second right intercostal space close to the border of the sternum.	Middle of sternum—also heard over aorta and carotids.	Regurgitation of blood through aortic orifice into left ventricle.
Mitral Orifice—Diastolic Murmur. (Mitral obstruction.)	At the apex or just at the position of the apex beat.	Usually limited to the apex beat. Rarely diffused.	Obstruction to flow of blood from the left auricle to left ventricle.
Pulmonary Orifice—Diastolic Murmur (Pulmonary regurgitation.)	Second left intercostal space close to the border of the sternum.	Transmitted down the sternum. Only to be distinguished from aortic regurgitation by pulse.	Regurgitation of blood through pulmonary orifice into right ventricle.
Tricuspid Orifice—Diastolic Murmur. (Tricuspid Stenosis.)	Median line of sternum on a level with fifth intercostal cartilage.	Base of heart or to the right axilla.	Obstruction to flow of blood from right auricle into right ventricle.

*[Modified from Manual of Clinical Diagnosis by Dr. Albert Abrams, 1894.]

guishing pericardial or exocardial from endocardial murmurs:

1. Unlike endocardial murmurs which are limited to a certain phase of the heart's action, they might be systolic, diastolic, or both, or even presystolic.

2. They are increased in intensity upon pressure with the stethoscope, which maneuver facilitates the friction between the pericardial layers.

3. During inspiration the lung approximates the layers of the pericardium, thus increasing during this phase of respiration, pericardial murmurs. Endocardial murmurs by the same act are diminished in intensity, because the interposed lung offers a poor medium of conduction to the chest wall.

4. The closer the two layers of the pericardium are approximated, the louder the murmur. To facilitate this approximation I would suggest pressure being made in the *intercostal spaces* and not on the ribs, as is the conventional practice. The same maneuver is applicable in the elicitation of pleural friction sounds. To make pressure with the stethoscope in the intercostal space, a phonendoscopic attachment may be fitted to the chest piece of any stethoscope according to the illustration. A piece of tin may be easily fitted by any tinsmith. In the center of the tin a rod terminating in a small button is screwed.

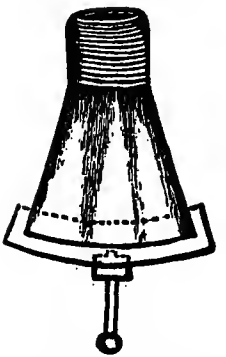


Fig. 4—Dr. Abrams' Modified Stethoscope.

5. They are circumscribed and are not transmitted beyond the area of cardiac dulness.

6. Change of position exerts a greater influence on the character of pericardial than endocardial murmurs. The former are especially distinct when the patient is in the sitting posture, with the body inclined to the left side.

7. They give the impression of being superficial in origin.

8. They frequently change their character, whereas the character of endocardial murmurs is almost constant.

9. They are rough, grating to and fro, or rubbing and scratching sounds.

10. When doubt arises whether a murmur is peri or endocardial in origin always remember that organic endocardial murmurs modify the

pulse and induce secondary effects upon the muscle of the heart.

PLEURO-PERICARDIAL MURMURS.

These murmurs often simulate pericardial murmurs. They arise when the pleura or peritoneum adjacent to the heart is roughened. They are modified by respiratory movement, disappearing or diminishing when the breathing is suspended or disappearing after forced expiration. Deep inspiration will usually accentuate them.

CARDIO-RESPIRATORY MURMURS.

These are sounds synchronous with the heart's action, produced outside this organ and heard usually to the left of the apex beat. Two factors enter into the production of these murmurs. 1. Forcible expulsion of air from the lungs by the heart striking against it. 2. With each cardiac contraction the bulk of the heart is reduced in size and a corresponding vacuum produced in the chest, which the lung compensates by expanding, thus producing a murmur.

ANEMIC MURMURS.

In anemia murmurs are frequently heard over the heart and vessels. They are endowed with certain characteristics: 1. They are soft and blowing in character and not prolonged. 2. They are systolic in time. 3. Generally loudest at the base of the heart and especially over the pulmonary orifice, a point where organic systolic murmurs are

often heard. The chief means of differentiation between the two lies in the fact that with organic we find dilatation and hypertrophy of the heart which are usually absent in anemic murmurs. 4. They are unaccompanied by changes in the size of the heart. 5. They frequently change their character. 6. They are accompanied by anemic symptoms and murmurs in the veins of the neck. 7. They are louder in the recumbent than in the upright position. 8. They are not transmitted away from the heart. 9. Under appropriate treatment with chalybeates they can be made to disappear.

PULMONARY ANEMIA.

I have described (Medical Standard, Jan. 1900) an *anemia of pulmonary origin,* in which anemic murmurs are frequent. In this form of anemia the ferruginous preparations are without effect on the murmurs which only yield to systematic lung development, inasmuch as the cause of pulmonary anemia is dependent on collapsed areas of lung.

HEMIC MURMURS IN THE LARGER ARTERIES.

The normal systolic and diastolic heart sounds are heard in the carotid and subclavian arteries. Pressure with the stethoscope over one of the large arteries will create a systolic murmur. Murmurs from the heart are often propagated to the large arteries. Of all the arterial murmurs likely to

perplex the physician, the subclavian murmur is the most frequent. It is regarded by many clinicians as a sign of phthisis. From an investigation of more than 300 cases (Vide my paper Medical Standard, Oct., 1899), I am able to formulate the following conclusions:

SUBCLAVIAN MURMUR.

1. The subclavian arterial murmur is an independent and rarely a transmitted murmur.

2. Its point of maximum intensity is the fossa of Mohrenheim, with feeble tendency to propagation. (The fossa is a depression under the clavicle in the outer part of the infraclavicular region between the pectoralis major and deltoid muscles.)

3. It is heard most often on the left side, less frequently on both sides and least frequently on the right side. In order of frequency it is heard at the height of inspiration, at the end of expiration and after momentary suspension of respiration.

4. It is usually a succession of murmurs uniform in character and intensified by certain maneuvers, notably deep inspiration, suspension of respiration and voluntary stretching of the neck.

5. One of its chief characteristics is its momentary duration, disappearing usually after a few deep inspirations.

6. Its dependence on the phases of respiration distinguishes it from all transmitted murmurs.

7. It may be present at one and absent at a subsequent examination, and neither its character nor duration is ever uniform from one examination to another.

8. The position of the patient may influence its genesis, but this is never sufficiently uniform to be of practical value.

9. A phthisical lung is not specially propitious to its occurrence, as it is found nearly as often in healthy as in phthisical persons.

10. It was present in thirty-six per cent of all healthy persons examined, advantage being taken in this enumeration or re-examination and those propitious factors which determine its occurrence, viz.: respiration and decubitus.

11. The *venous subclavian* murmur was only heard in six individuals with a preponderance of its occurrence on the right side.

12. The arterial subclavian murmur could be artificially induced on the left side in nearly 80 per cent of all individuals examined, and on the right side in about 65 per cent of the cases by a simple maneuver, viz., raising the arm gradually until it assumes a vertical position, while auscultating the Mohrenheim fossa during the time that the arm is brought to the latter position, the mur-

mur suddenly appearing at some time during the execution of the movement.

13. By the foregoing maneuver the subclavian venous murmur could be induced on the right side in 43 per cent of all persons examined.

DIAGNOSIS OF ENLARGEMENT OF THE HEART.

Thickening of the muscular walls of the heart is known as *hypertrophy*, while enlargement of one or more chambers of the organ is known as *dilatation*.

HYPERTROPHY OF THE HEART.

In hypertrophy, the left ventricle is most frequently involved owing to the increased work put on it by valvular lesions, diseases of the blood vessels, muscular exertion, etc. Its fellow ventricle on the right side hypertrophies in valvular lesions and in lung diseases whenever there is obstruction to the blood flow through the pulmonary organs, or, as we often say, increased resistance in the pulmonary circulation.

The symptoms of hypertrophy of the left ventricle are those of increased tension in the arterial system, viz.: congestive headaches, noises in the ears, and flushing of the face. The physical signs of the increased tension are: forcible and heavy heart impulse, the first sound at the apex is dull and prolonged while the second aortic tone is accentuated. The sounds are of course modified if valvular lesions are present. The pulse is reg-

ular, full, strong and of high tension. In hypertrophy of the right ventricle, increased tension may be manifested by hemoptysis owing to rupture of the blood vessels. Reliance, however, must be made on the objective examination. Over the tricuspid area, the first tone is louder and more prolonged than normal, while the second pulmonic tone is accentuated.

Hypertrophy is usually attended by dilatation, hence in left ventricle hypertrophy, the apex beat instead of being felt in the fifth interspace, two inches below and one inch to the right of the left nipple, is felt in the sixth, seventh or eight interspace, from one to three inches outside the nipple. Percussion shows increased dulness upward and transversely. If dilatation attends an hypertrophied right ventricle we find, bulging of the lower part of sternum, dislocation of the apex beat to the left, but rarely displaced downward. A marked epigastric impulse is noted in the angle between the ensiform cartilage and the seventh rib. The percussional area of dulness is increased transversely toward the right.

DILATATION OF THE HEART.

Dilatation of the heart is an evidence of weakness of the organ and it usually follows hypertrophy. It is the very earliest evidence of compensation failure. The symptoms are the reverse of hypertrophy, because the ventricles are incap-

able of emptying themselves at each systole. The apex beat is of course dislocated when the left side is involved, but it is very feeble and not punctuated, as in hypertrophy, but diffused. When the right ventricle is dilated, the impulse is seen and felt to the right of the ensiform cartilage. The action of the heart is irregular and intermittent. The heart tones are feeble and assume a fetal heart rhythm (embryocardia), i. e., the first and second heart sounds are alike and the long pause is shortened.

THE PULSE IN HEART DISEASE.

In palpating the pulse we must take into consideration: 1. Condition of the arterial wall. 2. Tension or blood pressure. 3. Volume. 4. Rhythm. 5. Frequency.

CONDITION OF ARTERIAL WALL.

1. In health the radial artery can easily be compressed and distinguished from other tissues. In *atheroma* of the arterial system, it is with difficulty compressed and may be rolled like a cord or pipe stem. Atheroma or arterio-sclerosis is a senile phenomenon and illustrates the fact, that the duration of life is decided by the condition of the arteries or, axiomatically expressed, "A man is only as old as his arteries." Alcohol, lead, gout, syphilis and other intoxications are common causes. Atheroma by increasing the blood pressure results in hypertrophy of the left ventricle

and the latter sign associated with a high tension

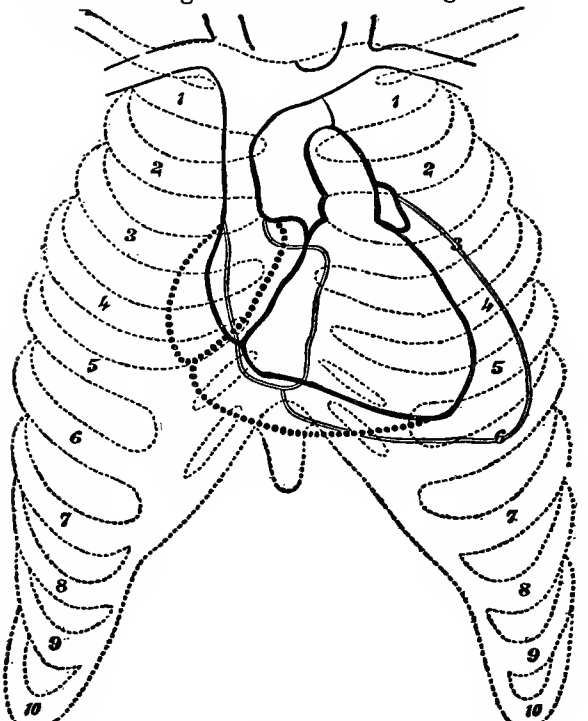


Fig. 5.

Diagram to illustrate the effect of dilatation of the right and left sides of heart respectively (Gee after v. Dusch). Continuous heavy outline, normal heart; dotted line, dilatation of right side; thin double line, dilation of left side.

pulse and accentuation of the second aortic sound are pathognomic of *arterio-sclerosis*. Angina pectoris owing to atheromatous involvement of the coronary arteries is common in arterio-sclerosis.

TENSION OF THE PULSE.

2. The pressure with which the blood flows in the arteries depends upon the degree of peripheral resistance and the force of the ventricular contraction. Normally, the pulse almost subsides between the beats, but little pressure being required to obliterate it. When the tension is increased, the artery remains continuously full between the beats. A pulse of low tension is soft and very compressible. It is indicative of heart weakness.

VOLUME OF THE PULSE.

3. This is dependent on the amount of blood in the artery; therefore in aortic and mitral stenosis the volume is small.

PULSE RHYTHM.

4. Disturbance of rhythm is manifested by intermission or irregularity of the pulse beats. Intermission means a dropping of a pulse beat and may occur at regular or irregular intervals. An intermittent pulse is characteristic of a fatty heart, if associated with a weakened first heart sound and evidence of failing circulation (edema of the feet). It is a symptom of coffee, tobacco, tea or digitalis intoxication. An irregular pulse is evidenced by

differences in time, force or volume of successive pulse beats and is of more serious import than an intermittent pulse. It occurs in mitral lesions and cardiac degeneration.

FREQUENCY OF THE PULSE.

5. In nearly all valvular heart lesions, excepting aortic obstruction with failing compensation, the pulse may be increased in frequency. Vagus disease and heart weakness are associated with an increased pulse rate. Diminished frequency of the pulse rate (*bradycardia*) may be associated with certain forms of cardiac disease, especially aortic obstruction. Appearing late in valvular lesions, it is usually an ominous sign.

The *sphygmograph* is an instrument of refinement to the practical physician in as much as palpation alone will detect all the variations in the pulse.

RECAPITULATION.

Mitral Insufficiency.—Pulse is small and feeble because the arterial system is devoid of blood.

Mitral Stenosis.—Pulse small and irregular with increased frequency.

Aortic Insufficiency.—Rapid recedence of the pulse as it strikes the finger (Corrigan's Pulse), especially if arm is elevated.

Aortic Stenosis.—On account of obstruction to the flow of blood, the left ventricle is hypertro-

phied, hence the pulse is one of high tension but lessened in volume.

Myocarditis.—Pulse small, soft and irregular; frequency, normal, diminished or increased.

A comparatively strong pulse, with feeble apex beat and heart tones is of great value in the diagnosis of exudative pericarditis. The strength of the right ventricle should never be gauged by the pulse, the loudness of the second pulmonic tone should be the index of its vigor.

Measuring the Intensity of the Heart Tones.

We are unfortunately in possession of no accurate means of registering the heart tones to facilitate accuracy in determining the progress of patients with heart lesions, or the action of cardiotonics. I have already reported (Medical News, July 8, 1899) the following method, which is only relatively accurate:

It is based on the simple physical principle that the intensity of sound varies inversely as the square of the distance from the sounding body, hence the distance to which a heart sound may be heard depends upon its intensity, ignoring of course those adventitious causes of propitious conductivity. Between the area auscultated and the stethoscope a medium is interposed. Experiment has taught me that one of the best media is partially vulcanized rubber in the form of a rod, and

just sufficiently soft as not to interfere with convenient manipulation. Such rods may be purchased in any store where rubber goods are sold. The circumference of the rods must equal the caliber of the pectoral end of the stethoscope in which they are to be inserted. The degree of insertion must be regulated by a notch cut into the rubber. The object of this regulation is to insure uniformity of results in the examination of individual patients. The rods may be of different sizes, varying in length from 6 to 26 centimeters, or even of greater length.

Before auscultating the heart tones by this method, we must first mark on the chest the different points in the precordial region, where the

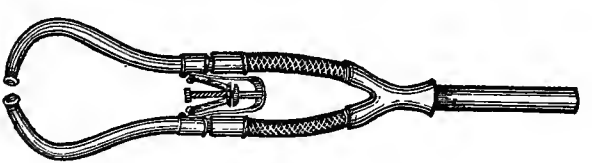


Fig. 6.

Rod inserted into the pectoral extremity of the stethoscope for measuring the intensity of the heart tones.

heart tones are heard with the maximum degree of intensity. Over each ostium we auscultate with the rod inserted into the end of the stethoscope, beginning with a rod of medium length and gradually increasing the length of the rod until one is

attained through which the heart tones are no longer conducted. The tubes are numbered, and a record may be made in our case book after the following formula.

Mitral, I tone.....6
Mitral, II tone.....5
Aortic, I tone.....4
Aortic, II tone.....5
Tricuspid, I tone.....6
Tricuspid, II tone.....4
Pulmonary, I tone.....4
Pulmonary, II tone.....5

According to the foregoing formula we conclude the following: That with a rod (No. 6) which is 26 centimeters in length we may still be able to hear the following tones: Mitral systolic and tricuspid systolic tones. A similar interpretation may be deduced from the other numbers. These figures possess no value for general application as the degree of transmission is dependent on the character of the stethoscope as well as the length of the rod employed. Each observer must cut his own rods of different lengths. With some kinds of stethoscopes the first mitral and tricuspid tones are still heard with rods fully 30 centimeters in length, whereas with other kinds a rod of half the length will no longer transmit the same tones.

In some instances another method may be adopted. It is less reliable than the former method, especially in thin persons, owing to the

increased conductivity of the thoracic tissues. As before, one marks on the chest wall the different situations where the heart tones, corresponding to each ostium, are heard loudest, and then proceeds in different directions until the sounds are no longer audible. The distance to which the sounds are propagated is marked and measured. The directions in which the sounds are auscultated have been determined empirically as follows:

MITRAL TONES.—Auscultate along a line on a level with the apex-beat to the left axillary region.

TRICUSPID TONES.—Auscultate along a line extending from the point of auscultation to the right axillary region.

AORTIC TONES.—Along a line on a level with the point of auscultation to the right axillary region.

PULMONIC TONES.—From the point of auscultation to the left axillary region. The tricuspid and mitral tones are best conducted downward by the liver, but as a differentiation of the mitral and tricuspid tones over the hepatic region is impossible this direction cannot be employed. I will mention, parenthetically, that the liver is an excellent conductor of the heart tones, and when they are no longer audible by auscultation we can safely conclude that the lower border of the liver has been reached.

Inhibition of the Heart as an Aid in Diagnosis.

The inhibitory nerve of the heart is the vagus, stimulation of which stops the heart in diastole. Czermak was able to press his vagus nerve against a little bony tumor in the neck, and by thus subjecting the nerve to mechanical stimulation was able to slow or even stop the beating of his own heart. If, in almost any healthy person, the carotid artery, or a point immediately adjacent to it in the neck, is compressed, slowing or complete inhibition of the heart and pulse ensues. This phenomenon is explained by compression of the vagus lying alongside the carotid artery.

Friedreich, and subsequently Sewell of Denver, observed that strong pressure with the stethoscope on the chest could cause the disappearance of murmurs, especially in individuals with an elastic thorax, which was attributed to inhibition of the heart movements.

I have endeavored to employ the phenomenon of cardiac inhibition as an aid in diagnosis. Observation has taught me that, for clinical purposes, inhibition of the heart is best attained by forcible voluntary contraction of the muscles of the neck. In some instances, the inhibitory effect on the heart is best observed when the head is stretched backward, and, when in this position, contraction of the neck muscles is attempted. With some per-

sons, to whom no instructions are intelligible, I place a long narrow cushion on the front of the neck and then ask them to press with all their might on the cushion with their chin. If too much violence is used in any of these maneuvers, the primary effect will be to increase the rapidity of the heart.

If the maneuver is properly executed, we diminish the intensity of cardiac tones and murmurs, and it is this fact that determines the real value of cardiac inhibition in diagnosis. A few seconds

Fig. 1—Normal pulse.

Fig. 2—Pulse during cardiac inhibition.

usually elapse before the effect on the heart becomes manifest, then, while the subject is still forcibly contracting the muscles of the neck, the heart tones become less and less evident, assuming an embryocardial character, until finally they are no longer audible. The accompanying sphygmogram was obtained from an individual on whom the method was tried for the first time.

We note almost total annihilation of the pulse

after irritation of the vagus by the contracted neck muscles. My investigations with this maneuver may in brief be summarized as follows:

1. Organic heart murmurs will become faint and often inaudible.

2. Transmitted murmurs are more amenable to the maneuver.

3. The fainter the murmur, the more easily it is suppressed.

4. When a transmitted murmur can be inhibited, the tone which it masks can be auscultated.

5. Heart tones are less amenable than murmurs to inhibition.

6. Hemic murmurs are more readily inhibited than organic murmurs.

7. When the murmurs of anemia are inhibited, they are replaced by tones.

8. Incorrect execution of the maneuver will intensify rather than diminish murmurs.

9. The inhibition maneuver when too often repeated is futile in its results owing to over stimulation of the vagi.

10. The maneuver enables us to determine the condition of the vagi as inhibitors of the heart and guides us in the administration of cardiotonics.

ILLUSTRATIVE CASES.

The value of the method is illustrated by the following cases:

1. Murmur audible during diastole in the second right interspace. At apex, systolic tone and diastolic murmur. During inhibition, the murmur in the second right interspace becomes fainter, while the diastolic murmur at the apex disappears and is replaced by a tone. Diagnosis: Aortic incompetency. The diastolic murmur at the apex is a transmitted murmur.

2. Loud murmur audible during diastole in the second right interspace. At the apex, systolic murmur and diastolic tone. During inhibition: Murmurs over aorta and apex persist but are less loud. Diagnosis: Aortic and mitral incompetency. The systolic murmur at the apex is not transmitted but is dependent on mitral incompetency.

3. Systolic murmurs over all the ostia and not transmitted away from the heart. Blood evidence of anemia. Inhibition: Systolic murmurs replaced by systolic tones. Diagnosis: Murmurs of anemia.

4. Systolic and diastolic murmurs at base of heart, modified by pressure with stethoscope and position of patient. Anemia not present. Inhibition: Murmurs disappear and replaced by tones. Diagnosis: Pericardial murmurs.

5. Murmur at fourth left interspace. Heart irregular, and rapid. No anemia nor sign of pericarditis. Inhibition: Murmur disappears to be replaced by a tone. Diagnosis: Cardio-muscular murmur.

The X-Ray in Cardiac Diagnosis.

A few years ago I exhibited before the California State Medical Society a series of lantern slides illustrating cardiac lesions diagnosed by the aid of the Roentgen rays. Many of my auditors no doubt regarded my exhibit as manufactured evidence, whereas others, less captious, were inclined to regard the demonstration as a joke. The vast amount of literature that has since accumulated has convinced the most skeptical that the Roentgen rays are invaluable in cardiac diagnosis. With the rays, we can accurately determine the size of the heart and learn in what part the organ is enlarged, and all this with more certainty than by any other method of examination. Aneurism of the heart may be accurately diagnosed, an impossible feat with other physical methods; aortic aneurism may be demonstrated even before subjective symptoms are experienced. By means of the Roentgen rays, we are enabled to gauge the action of digitalis and the Schott method of treatment on the heart with perfect ease. Pericardial effusion, dislocated, transposed and congenital malformations of the heart may be accurately de-

termined. For all this, two things are essential: Good apparatus and the services of an expert interpreter of skiascopic pictures. Without a Roentgen ray apparatus no physician can lay claims to scientific refinement in cardiac diagnosis.

CHAPTER III.

GENERAL TREATMENT OF THE DISEASES OF THE HEART.

I. Prevention. II. Treatment during compensation. III. Treatment during broken compensation. IV. Treatment of individual symptoms.

Prevention.

Acute articular rheumatism is one of the chief predisposing factors in the etiology of valvular lesions. We are constrained to heed the wise injunction of Sibson, that complete rest, during and after an attack of rheumatism lessens the average percentage of cases in which cardiac complications develop. We may profit by the experience of Chambers, who tells us, that during an attack of rheumatism, cardiac complications develop less often, when patients sleep in blankets and not between sheets. Sheets become wet with the acid perspiration and conduce to relapses from chilling of the skin.

The salicylates are almost specific for the arthritis, but they are not prophylactic against cardiac inflammation. The alkaline treatment according to Garrod, viz.: 40 grains of the bicarbon-

ate of potassium and 5 grains of citric acid, every 2 hours continuously until the urine becomes and remains alkaline and smaller doses thereafter, is the most certain means we possess for preventing and arresting heart complications. With the alkaline treatment the use of salicylates may be employed.

The gouty tendency is often associated with high blood tension, arterial degeneration and cardiac hypertrophy. Individuals showing this tendency must guard against over-feeding, indulgence in alcohol and live an open air life with an abundance of well regulated exercise. The inordinate use of alcohol is an important factor in etiology. Arterial degeneration and heart failure associated with dilatation of the organ are well recognized conditions in the inebriate.

Tobacco, like alcohol, must be interdicted in those who show a tendency to cardiac disease. Tobacco augments the cardiac contractions and induces intermittences and irregularities (arrythmia) of the heart. In the etiology of spurious angina pectoris, nicotine poisoning is paramount. An effective argument to induce tobacco habitues to discontinue their habit, is to instruct them to count the pulse before and after smoking, when they will invariably note an increase of from 4 to 11 beats a minute. Coffee and tea are not without influence in the etiology of

affections of the heart, notably, functional disturbances.

Syphilis is frequently concerned in endo-peri and myocardial lesions. Arterial syphilis is of common occurrence. Syphilitics, therefore, must be vigorously treated by inunctions or intravenous injections upon the advent of cardiac complications. Gonorrhea is frequently a factor in the etiology of endocarditis, gonococci having been frequently demonstrated on the implicated endocardium.

Moral hygiene is of importance in those predisposed to or suffering from heart disease. All emotions directly influence the heart and the epigram of Peter is worth repetition, "The physical heart is the counterpart of a moral heart."

Diet is of great moment in many functional heart affections. Food must be eaten in small quantities and be easy of digestion. Overloading the stomach, especially at night, must be avoided. Carbo-hydrates, owing to their tendency to form gases, must be used sparingly. Laxatives must be given to aid the abdominal functions. Digestive reflex neuroses of the heart are not infrequent after errors in diet. Dyspnea, palpitation and irregular heart, epigastric pulsation and psychic depression are a few of the symptoms following indigestion in some persons.

The effects of muscular strain on the heart

must not be forgotten, and occupations must be recommended which demand no excessive nor sudden muscular work nor exposure to cold and wet. Badly fed laborers often suffer from dilatation of the heart without valvular disease. In lifting heavy weights, such individuals, first take a deep inspiration and then suddenly stop expiration during the time severe exertion is made. The effect would be to empty the veins into the chambers of the heart leading to dilatation of the cavities. Prolonged rest should always follow heart strain, otherwise chronic irritability of the heart with dilatation ensues.

Treatment During the Stage of Compensation.

In the early history of medicine, patients with cardiac hypertrophy were made the subjects of a depleting treatment and they were placed on a low diet. Luckily for the patients, this error in therapeutics is no longer perpetrated. The province of the physician, during the stage, is strictly limited in maintaining the vigor of the heart muscle.

The great majority of those afflicted with compensated valvular lesions, suffer no inconvenience for years nor is the duration of their existence appreciably abridged. Clark, in 684 chronic valvular lesions which had been kept under observation for 5 years, noted no physical inconvenience

in any of the patients. Unfortunately, the belief yet survives, that the demonstration of a cardiac murmur, is the signal for digitalis, notwithstanding compensation is present. Hypertrophy of the heart, which is practically compensation, is an effort on the part of nature to overcome the circulatory disturbances resultant on valvular lesions.

Our efforts must be directed toward inviting hypertrophy and when present to encourage its existence. We must "make the heart equal to its task" (Beau). To maintain compensation the preceding remarks on prevention are germane.

The rules of prophylaxis can only be executed with the intelligent co-operation of the patient, who must be informed in a judicious way of the nature of his trouble. My almost invariable rule is to tell the patient that his trouble is purely a functional one, that unless certain laws of health are observed, it may become organic. The apothegm, "Ignorance is bliss," is especially applicable in the case of the cardiopath. "Hope springs eternal in the human breast" may refer to the phthisical, but never to the cardiac patient.

Systematic exercise must not be inhibited, on the contrary, it is now regarded as an invaluable aid in maintaining the muscular power of the heart and increasing it. The character of the exercise taken is of little moment, provided no dyspnea, heart distress or palpitation follows. The

slightest evidence of such symptoms is a signal of danger.

Provision by the usual preventive measures must be taken against catching cold. Every attack of bronchitis throws an additional burden on the heart. Climate is a valuable adjunct in treatment. Extremes in climate must be avoided. Mild temperate climates with cool weather are to be favored. High altitudes in general must be avoided. Observations teach us that it is the right heart which is first overtaxed by a sojourn in high altitudes and this observation applies with equal cogency to the healthy heart.

TREATMENT DURING FAILURE OF COMPENSATION.

Broken compensation asserts itself slowly. Among the earliest subjective symptoms are dyspnea on exertion, nocturnal paroxysms of dyspnea and cardiac distress. Objectively, small, irregular and feeble pulse and localized edema are characteristic. The chief object of treatment is to restore the enfeebled heart muscle which is attained by rest, the use of agents which stimulate the heart's action and by methods which relieve the embarrassed circulation, viz.: Venesection and depletion by purgation.

The heart receives two sets of nerves, the excitary from the sympathetic system and the moderator nerves derived from the pneumogastric. While the *excitatory* nerves put the heart muscle in

action, the moderator nerves inhibit the movements, but, by harmonious action of these opposite nerve influences, the regularity of the heart contractions is due.

Absolute rest in bed is one of the supreme triumphs of cardiac therapeutics. By this method alone, the relief of the symptoms of failing compensation is oftentimes phenomenal and but two or three weeks' rest usually suffice to attain the object. The rest must, however, be as absolute as in the rest cure method of Weir-Mitchell and the nourishment must be equally exacting. If anemia is present, the liberal use of some assimilable chalybeate is indicated. In addition, we must remember the great value of fresh air, sunshine and a cheerful environment. When rest in bed alone fails to restore the circulatory equilibrium, the recourse must be had to cardiac stimulants and tonics.

CARDIAC TONICS.

The sovereign heart tonic is *digitalis,* the quinine of the heart. Digitalis slows the action of the heart and increases the force of its beats; the blood pressure in the arterial system rises with contraction of the peripheral arteries. The physician is frequently bewildered in encountering in the text books, prolix and elaborate indications and contraindications for its use. An invariable indication for its use is dilatation of the heart,

stationary or progressive, irrespective of the nature of the valvular lesion. The physician unskilled in methods of cardiac percussion is justified in its use, in all cases of compensation failure. There are some authorities who declare that its use is dangerous in aortic incompetency, because by prolonging diastole it promotes the regurgitation of blood into the left ventricle. This objection is purely theoretic.

Some contraindications against its use are excessive slowing of the pulse present in some cases of idiopathic myocardial disease as well as in stenosis of the aortic and mitral orifices. The danger of arterial rupture, owing to the increased blood pressure which attends its physiologic action, I believe to be theoretical. Of one contraindication one can speak absolutely and that is, it should never be used when compensation is properly balanced.

When digitalis acts favorably, we note the following: Pulse becomes slower, regular and increased in tension. Dyspnea and dropsy disappear. The urine formerly scanty, high colored and depositing urates becomes light colored with diminished specific gravity and is very much increased in quantity. In the use of the drug we must always anticipate toxic symptoms which are gradual in their appearance, viz.: Nausea, vomiting, small irregular pulse and diminished excretion of urine.

These symptoms usually disappear when the drug is withdrawn and are rarely serious. Digitalis must be continued until compensation has been restored. During the course of its administration, it is well to suspend its use for a day or so in anticipation of its cumulative action. When nausea attends its use, it may be given by the rectum, preferably in the form of the infusion.

Digitalis has often been unjustly discredited as a drug, owing to many inert preparations found in the shops. The most reliable preparations are those secured from trustworthy eclectic and homeopathic pharmacists as they are in honor bound to use the fresh leaves. After curing, digitalis leaves rapidly deteriorate. Authorities are not in accord on the preparation to be employed. Some prefer the watery, others the alcoholic preparation. The two preparations are by no means identical in action, the glucosides (digitalin, digitoxin, etc.) vary in solubility in alcohol and water. The watery preparation, the infusion, is more effectually diuretic whereas the tincture has a more direct influence on the heart. The glucosides ought not to be employed, as our present knowledge of their composition and physiologic action is very uncertain.

The tincture of digitalis is administered in 10 to 15 minim doses every 3 or 4 hours, the infusion in ½ ounce doses at the same intervals. To secure

the best results with digitalis, I am in the habit of giving the tincture before and the infusion after meals. Osler voices the opinion of careful observers when he expresses the belief that there are no substitutes for digitalis.

Strophanthus. This is the only cardiac tonic which possesses any action similar to digitalis, but unlike the latter it is less reliable and energetic. Strophanthus increases arterial pressure by increasing the work of the heart, but unlike digitalis, it does not contract the blood vessels. It may be given continuously without fear of toxic manifestations, in fact, its action is only apparent after long continued use. In many instances the tonic effects on the heart initiated by digitalis may be continued with strophanthus which is usually given in from 5 to 10 drop doses 3 or 4 times a day.

Caffeine is regarded by some as almost equal to that of digitalis in diseases of the heart. It causes the beats of the heart to become stronger and occasionally more rhythmical. Unlike digitalis and strophanthus it has no specific action on the inhibitory nerves of the heart. Caffeine is frequently of service in cardiac disease when other cardiac tonics have failed to give relief and it is of especial value in cardiac dropsy alone or combined with digitalis. Caffeine is given in doses of from 3 to 5 grains, 3 or 4 times daily as the natrobenzoate

or natrosalicylate owing to their increased solubility and more rapid action.

Strychnin is a most efficient heart stimulant in sudden heart failure. By the mouth, in the doses usually recommended, I have seen very little effect. It must be given hypodermically in doses varying from 1-30 to 1-15 of a grain and frequently repeated. Lately other cardiac tonics have been recommended, but they are of subordinate value. They may be briefly referred to:

Spartein. Serviceable in valvular disease when dropsy is present. Dose, gr. 1-6 to ¼ every 4 to 6 hours.

Convallaria Majalis (lily of the valley). Effects on the circulation like that of digitalis, but less powerful and decidedly more uncertain. The best preparation is the infusion, in doses of from 2 to 8 drachms.

Adonis. An uncertain cardiac stimulant with marked diuretic powers giving it a supposed value in dropsy and fatty heart. Dose of the infusion, a tablespoonful, 3 or 4 times a day.

Nitro-glycerine. Cardiac stimulant and arterial relaxant. Useful in aortic valvular lesions when the object is to give relief to the violently acting left ventricle by dilating the peripheral blood vessels. Dose, one minim three times a day of the one per cent solution and increasing the dose one

minim each day until flushing or headache is experienced.

Cocain. Similar in action to strychnin. Dose, ¼ grain every 4 hours. The following tabular review will recall the essential facts necessary in the administration of cardiac tonics.

THE SCHOTT METHODS BY SALINE BATHS AND RESISTED MOVEMENTS.

These methods produce phenomenal results in overcoming the symptoms of disturbed compensation even after rest, digitalis and other cardiac tonics have failed. By these methods, the results achieved are due practically to, (1,) the removal of peripheral resistance which increases the arterial circulation; (2,) relief of venous congestion owing to the increased quantity of blood in the arteries; (3,) diminished work of the heart owing to free circulation of blood in the arterial system.

The Schott treatment is indicated in all functional disturbances of the heart and in valvular lesions complicated by incompensation. It is contraindicated in aneurism, chronic myocarditis and marked arterio-sclerosis. For more than 40 years the brothers Schott in Nauheim, Germany, have been active in the treatment of cardiac diseases by gymnastics and baths, but it is only in recent years that the Schott treatment has been revived

TABLE OF CARDIAC TONICS.

	DIGITALIS.	STROPHANTHUS.	CAFFEINE.	STRYCHNINE.	NITROGLYCERINE.
Physiologic action.	Stimulation of heart muscle and inhibitory apparatus; contraction of arterioles. Slows heart and increases force of its contractions.	Similar to digitalis, but does not contract arterioles. Less rapid than digitalis in action and less certain and powerful.	Increases the power and regulates the rhythm of heart, but without specific action on the inhibitory nerves. Acts through the nervous system.	Stimulation of the motor and inhibitory apparatus of the heart with rise of arterial tension.	Vascular dilation and consequent lowering of blood pressure. Increases action of the heart by removing influence of inhibitory apparatus.
Toxic symptoms	Nausea, vomiting; slow, irregular pulse; syncope, confusion of vision; insomnia, delirium. A blue sclerotic considered diagnostic. (Tardieu.)	Not cumulative in action like digitalis and more easily tolerated by the stomach.	In too large doses, over excites the nervous and circulatory systems, inducing insomnia and even mania.	Dilated pupils, spasmodic respiration, limbs jerk, stiff lower jaw and exaltation of all the reflexes.	Intense frontal headache, beating carotids, and rapid and irregular action of the heart.
Indications for use	Failure of compensation irrespective of the nature of valvular lesion.	Same as digitalis. May act when the latter fails. May be useful in aortic incompetency and arterio-sclerosis.	When rapid cardiac stimulation is necessary and as an adjuvant to other tonics when diuresis is necessary.	Powerful adjunct to digitalis; especially valuable in failure of right heart. Very reliable in sudden heart failure.	In painful angina and syncope occurring in aortic lesions.
Contra-indications	When compensation established. Theoretically, its use is interdicted in aortic incompetency and arterio-sclerosis.	None.	In irregular heart and when digitalis and strophanthus have not previously been tried.	Feeble in action when given *per orem*.	In arterio-sclerosis and irregularity of the heart.
Most efficient preparation and dose	Infusion in ½ oz. and tincture in 10 min. doses 3 or 4 times a day; continued until symptoms relieved; then replaced by strophanthus.	Tincture, min. 5-10, 3 or 4 times a day.	Caffein, benz. soda, each, 4.0 gm; dis. water, 150.0 gm; licorice, 30.0 gm; M. S. ½ to 1 tablespoonful 3 to 6 times aday.	Tr. nux vom. min. 5-10 and strychnine sulph. hypodermically 1-30 to 1-15 gr.	One min. of the one per cent. solution every hour until physiologic effects occur.

in interest. The methods consist in baths and resisted movements.

THE BATHS.

In this country, we are constrained to use artificial Nauheim baths. While I do not underestimate the value of the natural baths at Nauheim, I do not consider them absolutely essential. Three of my patients, who have taken the baths at Nauheim and the artificial baths at home, claim that in effects, there is absolutely no difference between the natural and artificial baths. I am inclined to believe that the real benefit from the baths is dependent on the temperature of the water and the generation of carbonic acid gas. I pursue the following method, disregarding the minutiæ, which are of no practical importance:

In 40 gallons of water, the amount usually necessary for body immersion, the temperature of which must be 95° F., 1 pound of sodium bicarbonate is dissolved. After the patient is immersed in the bath, 1½ pounds of hydrochloric acid (25%) is introduced in a bottle at the lower end of the bath tub, which must of course be of porcelain to avoid the action of the acid. Gradually the acid is poured from the bottle, resulting in the formation of carbonic acid gas. The patient remains in the bath for 15 minutes on an average, during which time he must remain absolutely quiet. Baths are given daily for 3 consecutive days and

then omitted on the fourth day, or about 21 baths in one month. The effects observed after the baths are almost immediate, viz.: lowering of pulse rate and increased strength, relief of cyanosis and dyspnea, marked reduction in cardiac area and a feeling of exhilaration.

RESISTED MOVEMENTS.

These are regular voluntary movements that the patient makes which are resisted by the operator. The movements are simply flexion, extension, adduction, abduction and rotation of the limbs, neck and trunk. Each single or combined movement is followed by an interval of rest. Patients must breathe regularly and uninterruptedly during the movements. The movements should be gentle and must at once be suspended should the patient show weariness or any increase in the number of respirations or any material increase in the number of pulse beats. The same muscles should not be exercised twice in succession. The duration of each sitting should at the beginning not exceed 10 minutes, and after the patient has become accustomed to the movements, 30 minutes is usually the time limit.

The baths give more permanent effects than the movements, whereas a combination of both methods yields the best results. When both are used,

the movements are given in the morning and the baths at night.*

In explanation of the reduction of the size of the heart and the good effects observed after the Schott treatment, I have espoused the theory (The Medical News, Jan. 7, 1899) that the baths and movements act by reflex stimulation through the skin. What I have called the *heart reflex* (Philadelphia Med. Journal, Jan., 1900) is a contraction of the heart muscle upon application of a cutaneous irritant (vigorous rubbing of the skin or a spray of ether to the precardial region). This contraction of the myocardium is easily demonstrated, especially in children by means of the Roentgen rays and the fluoroscope. Vigorous cutaneous friction will therefore reflexly induce contraction of the heart muscle.

The physiologic opinion has been gaining ground that the heart muscle is itself essentially motor, containing in its vital qualities the essential principles of its own activity and not depending for its action upon its nervous mechanism. Embryology furnishes one of the best proofs of this hypothesis, viz.: that the heart beats in the em-

* The Triton Company in New York has prepared salts for sale corresponding to the Nauheim Salts. They furnish a box containing sodium bicarbonate and 8 cakes of sodium bisulphate, the carbonic acid gas being generated by the action of these 2 salts upon each other.

bryo long before any nerve influence or fibres can be demonstrated in its substance. In many of my patients to whom the baths and the resisted movements are inconvenient, I have employed vigorous cutaneous friction with rough towels with most excellent results. As a rule, I initiate the frictions, after the patient is immersed in a bath (95° F.) for about 10 minutes.

LUNG GYMNASTICS.

Twelve years ago (Sacramento Med. Times, Sept., 1888) I urgently recommended pneumatic differentiation by means of the pneumatic cabinet as one of the most efficient agents then at our command, in overcoming the symptoms of cardiac failure, especially those dependent on an embarrassed pulmonary circulation. Time has in no wise moderated my views. The disadvantages attending this method are the cost of a pneumatic cabinet and the difficulty of its transportation. Results nearly as good may be attained by breathing exercises, systematically and persistently pursued. So competent an authority as Quimby (Boston Med. and Surg. Journal, Aug. 31, 1899) avers, "There is no therapeutic measure (referring to valvular lesions) whose action is so definite or constant."

The heart, like any other muscle, owes its vigor to the activity of respiration. The exceptional muscular strength of insects is no doubt due to the fact that they respire from nearly every part of

their bodies. Individuals with organic heart disease enjoy the best health when they are able to live in open air life. The principles of the "open air method" in the treatment of phthisis are equally applicable in organic heart disease. The excellent therapeutic results with iron in organic heart disease depend no doubt on the amount of oxygen conveyed to the tissues. As a prophylactic against myocardial degeneration, the value of an assimilable iron preparation cannot be praised too highly. Owing to the negative intra-thoracic pressure occurring during inspiration, the blood is facilitated in its flow to the chest and the effect is enhanced, the deeper the respiratory movement. Outside of the pneumatic cabinet, I know of no more efficient lung exercise than systematic voluntary forced inspirations and expirations, the movements of the thorax being unrestrained by clothing.

I have already reported (Medical Fortnightly, Sept., 1899) the results of my investigations with different methods and different apparatus in lung development. This was done while the Roentgen rays were traversing the thorax, the index of lung inflation, being the bright reflex as seen with the fluorescent screen. The investigations in brief demonstrated most emphatically that deep voluntary inspirations and expirations secured the most thorough lung inflation.

METHOD OF OERTEL.

This method aims in strengthening the heart muscle by exercise, diet and limitation of the ingestion of fluids. It is especially applicable in the treatment of "fat heart." The exercise is begun by directing the patient to walk on level ground a definite distance. The appearance of fatigue, dyspnea, or heart symptoms, indicates the degree of toleration, when walking is suspended and the patient must rest. It is advisable to instruct the patient to walk on some thoroughfare traversed by a street car, thus enabling the patient to ride home on the advent of fatigue. On the following day, the distance in walking is to be increased until finally a walk of a mile or two can be taken each day without inconvenience. Later, the patient, under the same precautions, is instructed to climb hills, climbing a certain distance each day, until, eventually, the top of the hill is attained without sense of fatigue. The diet is practically that which is applied in the treatment of obesity. The quantity of fluids taken must be diminished and the tissue fluids must be eliminated by exercise and sweat baths.

HOME EXERCISE.

When the Nauheim or Oertel methods cannot be conveniently taken, home exercise by means of springs or pulleys in which resistance can be accurately gauged may be recommended, always ac-

companying advice with the injunction, that exercise must always stop short of fatigue or heart distress.

Treatment of Individual Symptoms.

1. Palpitation. 2. Dyspnea. 3. Dropsy. 4. Cough. 5. Hemoptysis. 6. Nervous Symptoms. 7. Gastric Complications. 8. Renal Complications.

PALPITATION.

Relief should be attempted by the application of an ice-bag over the heart. At the same time, bromide of potassium may be given in 30-grain doses every 4 hours until relief is obtained. The latter drug has often a phenomenal regulatory influence on the heart and circulation, and its action is evidenced by the rapid reduction in the number of pulse beats. It also combats the nervous irritability so frequent in cardiac patients. Tincture of aconite (U. S. P.) in 1 to 3 minim doses every 3 hours, carefully watching its effects, is often of great value. Under its influence, the heart-beats become greatly reduced in number and power, the pulse slow, irregular and weak. Aconite is of undoubted value in functional cardiac disturbances, but when the heart is weak it must be used with circumspection, or better not at all. The further treatment of this symptom will be discussed under the treatment of special diseases.

DYSPNEA.

Here treatment must be directed to the cause: cardiac dilatation, bronchitis, pulmonary congestion and hydrothorax. The latter complication is frequently overlooked in cardiac dyspnea. Dyspnea of a paroxysmal character is practically nought else but cardiac asthma, for which amyl nitrite inhalations or nitro-glycerin internally may prove of service. When everything else fails, reliance can always be placed on satisfactory doses of morphin given hypodermically. Inhalation of oxygen as a palliative measure may be tried, but unless speedily effective, it is useless.

DROPSY.

Beside the usual cardiac tonics which augment the resorption of fluids, recourse must be had to diuretics, purgatives and sudorifics. We must never forget that cardiac dropsy always offers an increased resistance to the heart, and must therefore be gotten rid of as soon as possible. Cardiac asthma and lung edema are often marvelously relieved by agents which cause a resorption of the edematous fluid, digitalis fulfills the double function of cardiac tonic and diuretic. I make frequent use of the following formula:

Infusion of digitalis.....8 ounces.
Diuretin .....4 drams.

A tablespoonful three times a day for an adult.

A combination of strychnin, digitalis, spartein,

squill and caffein will often augment diuresis. Another excellent combination is the following:

Acetate of potash.....8 drams.
Infusion of digitalis.....8 ounces.

A tablespoonful three times a day for an adult.

Trousseau's diuretic wine is often useful:

Bruised juniper berries.....10 drams.
Powdered digitalis ..... 2 drams.
Powdered squill ..... 1 dram.
Sherry wine ..... 1 pint.

Macerate for four days and add:

Potassium acetate ..... 3 drams.

Press and filter.

A tablespoonful three times a day for an adult.

Calomel often proves to be an excellent diuretic in cardiac dropsy, even when digitalis fails. During its use, the excretion of urine becomes very large. When calomel fails in its action, we must be on the lookout for mercurialism.

Calomel is given in 2 or 3 grain doses combined with opium (gr. 1-6), 3 times a day. The addition of the latter is to overcome the tendency to diarrhea. Mercurialism is prevented by mouth hygiene. If at the end of five days increased diuresis does not occur, or if at any time during its use salivation arises, the drug must be suspended. The diuretic action of calomel is not usually manifest until the third day.

Galactotherapy.—Skimmed milk, 2 to 3 quarts daily is followed in a few days by augmented diu-

resis. If, after five days, the latter symptom is not manifest, it will usually fail. The ordinary diet must be taken in conjunction with the milk, as it is doubtful whether an exclusive milk diet can provide sufficient nourishment for an adult, a fact of great importance where nutrition is of such vital importance in the restoration broken compensation.

Purgatives.—The method of Hay is useful: Rochelle or Epsom salts (1 to 1½ oz.) in concentrated solution, taken one hour before breakfast, is followed by 3 to 6 watery evacuations daily. When salines fail and the heart is strong, drastic purgatives like the following may be used. Pulvis jalapæ comp. (3 gr. to 1 oz.), resina scammonii (5 to 10 gr.), extractum colocynthidis comp. (5 to 10 gr.), resina podophylli (1 to ¼ gr.), elaterin (Merck), (1-20 to 1-12 gr.).

Sudorifics.—Pilocarpine is the ideal diaphoretic, but on account of its deleterious action on the heart, should never be used. Instead, the hot bath, of 15 minutes' duration, after which the patient is wrapped in blankets, may be used. The hot air bath is often more convenient. The hot air may be conducted through a tube under the bedclothes raised under a low cradle. Sweat baths are usually well tolerated, although before using, the patient should be stimulated by whisky.

Relief of Dropsy by Surgical Means.—When

medicines fail, punctures through the skin to the subcutaneous tissue of the lower extremities should be made. A sterilized scalpel is usually employed for making the punctures, although a large-sized needle is equally useful. This method has fallen into disuse owing to wound infection following the punctures. To avoid infection, Southey suggested using fine silver trocars, with rubber tubes attached, so that the fluid could run off gradually. In this way, a few pints of edematous fluid may be disposed of in a day. After the incisions are made, I frequently employ a cupping glass to facilitate the removal of the fluid. Danger of infection is done away with entirely, if the physician conducts his minor surgery under the strict principles of asepsis. The skin to be punctured or incised is scrubbed and then washed with an antiseptic. Then with an aseptic scalpel, four small incisions are made on either side of the leg and immediately covered with borated cotton. The latter must be constantly renewed, when wet, by sterilized hands. With the patient in the sitting posture, the flow of fluid is greater. To facilitate the rapid removal of fluid, I often use the following method: Two incisions are made on either side of the thigh above the knee joint; then a Martin elastic bandage is applied beginning at the foot and extended upward to an inch below the incisions. The bandage forces the fluid toward the incisions.

COUGH.

This is a common symptom and frequently results from stasis in the pulmonary vessels with concomitant bronchial catarrh. Treatment directed toward incompensation is indicated. Codein may be tried, although heroin in tablets, 1-20 to 1-12 gr., several times a day has given me the best results.

HEMOPTYSIS.

This rarely calls for treatment. It is often a relief to the congested pulmonary vessels, and is rarely fatal. The all-important treatment when indicated is absolute rest in bed.

No faith is to be placed on the conventional hemostatics. The most reliance to be placed in the hypodermic use of morphin. Gelatin in solution introduced subcutaneously, may be tried. In a recent patient with intractable hemoptysis, large quantities of flavored gelatin taken by the mouth proved efficacious. A similar experience was had in two cases of purpura hemorrhagica.

NERVOUS SYMPTOMS.

For the insomnia and peculiar hallucinations of cardiopathic patients, paraldehyd and trional give excellent results. A dose of spirits of chloroform or ether in hot whisky will often give a quiet night. Chloral should not be used. Hydrotherapeutic measures may be tried, such as bathing the

face with cool water, an alcohol sponge or a wet pack with warm water. When everything else fails, morphin, hypodermically, may always be depended on.

GASTRIC COMPLICATIONS.

Stomach disturbances are oftentimes only relieved when compensation is restored. Until this occurs, little burden should be thrown on the stomach by careful dieting. A milk diet will often bridge over a period of gastric irritability. Starchy foods cause flatulency and must be proscribed. Concentrated meat extracts may be tried. They are easily absorbed, nutritious and stimulating to the heart. Of late I have used tropon, which represents over 90 per cent of pure albumin. It is insoluble in water and may be given in soup or with the yolk of an egg. It is not palatable.

RENAL COMPLICATIONS.

In renal complications, diet is of prime importance. Foods must be selected which are capable of easy digestion, and which are least liable to produce intestinal poisons and thus conduce to auto-intoxication. Arterial tension being high in these cases, nitrogenous food and fermented liquors should not be used. Pre-digested milk is the ideal food relieved by kumyss. A vegetable diet, excluding fibrous vegetables, such as turnips, beets, etc., and beans and asparagus, combined with fresh

fruits, is useful. When digitalis is used, it should be employed in conjunction with nitro-glycerin. The uric acid diathesis must be remembered as a common cause of high arterial tension, and the appropriate treatment must be directed toward the formation of uric acid and its excretion from the economy.

CHAPTER IV.

AFFECTIONS OF THE PERICARDIUM.

Acute Plastic or Fibrinous Pericarditis.

ETIOLOGY.

Rarely primary, as a result of traumatism. Usually secondary to the acute infectious diseases. Acute rheumatism is the chief etiologic factor in about 50 per cent of the cases. Especially in children, pericarditis may precede the joint symptoms. Next to the rheumatic, tuberculous pericarditis is the most frequent variety. The disease frequently complicates the septic processes. It may be one of the earliest symptoms of Bright's disease especially the interstitial form (pericardite Brightique, of the French). Gout, scurvy, cancer and leukemia are causes. From the contiguous tissues and organs, inflammation by extension may implicate the pericardium.

PATHOLOGY.

The exudation consists mainly of fibrin. Fluid may be present but never in large amounts. The superficial layers of the heart muscle may become implicated in the inflammatory process thus entailing cardiac asthenia which will gravely influence the prognosis.

SYMPTOMS.

No reliance must be placed on subjective symptoms, otherwise, the affection will, as it often is, be overlooked. Pain referred to the precordia or xiphoid cartilage may be present. The most trustworthy sign is the friction sound. It may be palpated but is more often heard. 1. It is a rubbing, scratching sound and appears to be quite superficial. 2. It is best heard over the right ventricle, the part of the heart approaching nearest the chest wall, viz., the fourth and fifth interspaces and neighboring parts of the sternum. 3. It is not, like the endocardial murmur, transmitted away from the heart 4. Its intensity varies with the position of the patient. 5. It is usually double, corresponding with both systole and diastole, but the synchronism with the heart tones is not absolute. One receives the impression that it is a superadded sound. I have frequently found that the rubber tip of the stethoscope will often create adventitious sounds not unlike the friction murmur. To obviate this error, my modified stethoscope illustrated in a previous chapter will be found useful. With it, one may make pressure in an intercostal space and thus accentuate the murmur to a marked degree. The ordinary phonendoscope is not available for such a purpose, as the least degree of pressure creates artificial sounds.

DIAGNOSIS.

For differentiation from other friction sounds, *vide* chapter on diagnosis.

COURSE AND TERMINATION.

Usually favorable to life. Rheumatic cases usually recover. The exudate may agglutinate the pericardial layers (*adhesive pericarditis*) or the plastic variety may be converted into a pericarditis with effusion.

TREATMENT.

Symptomatic and expectant. Routine measures are not justified. One is reminded of the story told of Sir Wm. Gull. At a consultation, the latter detected a pericarditis which had been overlooked. The attending physician was unduly apologetic for his oversight. Sir William replied, "Perhaps it is just as well you did not find it, for if you had, you might have treated it." Absolute rest in bed is generally demanded to reduce to a minimum the action of the heart. An ice bag to the precordia relieves pain and palpitation. Hot applications may prove more efficient. Blisters to the precordia, an old time practice, is not justified by modern knowledge. Their application interfere with a close study of the heart. Small doses of digitalis or strophanthus may be indicated to control the excited heart's action or when the pulse becomes irregular, intermittent and of low tension.

Pericarditis With Effusion.

ETIOLOGY.

A common sequence of the previous variety. About one-third of the cases are associated with acute rheumatism. Phthisis, septicemia and Bright's disease are among the etiologic factors. It may complicate the eruptive fevers or depend on an extension of inflammation from contiguous strictures.

PATHOLOGY.

The effusion is usually sero-fibrinous but may be hemorrhagic or purulent. The quantity of fluid may vary from six ounces to four pints. The pericardial layers are thickened and covered with fibrin. In favorable instances, absorption of the fluid occurs. As a rule, the fluid only is absorbed, the fibrinous exudate remaining to form adhesions between the visceral and parietal membranes. In the severe forms the superficial layer of the heart muscle beneath the visceral pericardium becomes functionally and anatomically involved. (*Peri-myocarditis.*)

SYMPTOMS.

No affection is more frequently overlooked. It may develop without symptoms. Pain and distress in the precordia may be the earliest symptoms. Pressure symptoms depend on the amount of the effusion.

Dyspnea or orthopnea is an early symptom of pressure.

Aphonia, due to compression of the recurrent laryngeal as it winds round the aorta, dysphagia, from pressure on the esophagus, irritative cough, from compression of the trachea, distension of the veins of the neck and compression of the left lung are other pressure signs. Altered cardiac rhythm due to the mechanic effects of the fluid on the heart interfering with its action is common. The pulse is rapid, intermittent and small. The paradoxical pulse may be present, i. e., a pulse in which the beats become weak or lost with each inspiration.

When the effusion is not large, a very important rational sign to remember is, that *the apex beat which is with difficulty palpated, may be associated with a comparatively strong pulse.*

The onset of the disease may be characterized by cerebral symptoms. The patient is delirious or may become melancholic and show suicidal tendencies. The condition may resemble delirium tremens. *The occurrence of delirium in acute rheumatic fever should at once direct attention to the heart.*

PHYSICAL SIGNS.

Inspection and Palpation. In young subjects, there is precordial prominence with obliteration and even bulging of the intercostal spaces. The apical beat is diffused or lost and if felt, is raised

and dislocated outward. Adhesions of pericardial origin may retain the apex to the chest wall despite the effusion. Ewart's sign, in which it is possible to feel the upper edge of the first rib together with its inspiratory and expiratory movements is regarded as trustworthy although it also occurs in some cases of heart dilatation.

Percussion. This is to be relied on most in diagnosis. The precordial figure of dullness is irregularly pear shaped; the base directed downward and the stem or apex directed toward the upper end of the sternum.

Sternal dullness is a suggestive sign. Normally the sternum is resonant owing to the contact of its upper part with the lungs. When this contact ceases to exist, as occurs in pericardial effusion when the lungs are separated from the sternum, percussion of the latter bone will yield dullness. This sign cannot be regarded as diagnostic because an enlarged heart may have the same effect on the lungs.

The Rotch sign is important in diagnosis. As a result of effusion within the right corner of the pericardial sac, the usually resonant area in question may become dull on percussion. This area is in the right fifth intercartilaginous space formed by the right border of the heart and right lobe of the liver (cardio-hepatic triangle). Dullness of the triangle has been observed, though rarely, in

cases of enormous dilatation of the right auricle from tricuspid stenosis.

Depression of the liver is more marked in pericardial effusions than in any other intra-thoracic affection, the possible exception being pneumothorax. The hepatic percussion note may begin at the level of the tip of the xiphoid instead of at the infra-sternal notch. As a result of the depression, the fingers applied below or at the side of the xiphoid can be made, by pushing upwards and backwards, to ride over the upper surface of the liver, which is normally out of reach.

The posterior pericardial patch of dullness in association with other symptoms furnishes a complete and crucial evidence of fluid. Whenever fluid accumulates in the pericardium, a marked patch of dullness is found at the left inner base, extending from the spine for varying distances outward.

The Respiratory Sign. I have designated this the respiratory sign because the area of precordial dullness is dependent on the amount of air in the lungs. Normally it is possible to obliterate the superficial area of cardiac dullness by deep inspiration. Even in extreme cases of cardiac dilatation, the area of heart dullness may be diminished by forced inspiration. In effusions, the influence of forced inspiration is extremely slight or absent.

Auscultation. The heart tones are feeble or dis-

tant and scarcely heard. The friction sound heard in the beginning may disappear but often persists at the base or perhaps at a limited area of the apex. An important sign, if the patient is seen early, is to note the diminishing loudness of the heart tones with increasing effusion.

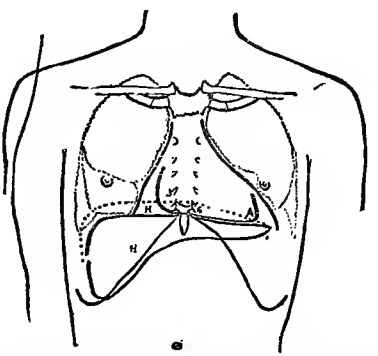


Fig. 9—Illustrating "Rotch's sign" (dullness in the right 5th space—5 to H); also contrasting the angle (on either side of H) of the dullness as due respectively to effusion and to dilatation. The heart's outline is normal in size and position. The outer lines are those of the dullness in moderate effusions. The "suprahepatic line" (dotted) and the "hepatic line" limit the normal "modified" dullness of the liver; and H is placed on the absolute dullness.—(Ewart.)

Bamberger's Sign. When the patient is sitting upright an area of dullness about the size of a silver dollar can be detected at the angle of the scapula. On auscultation of this area, tubular breathing is heard. If the patient leans forward,

dullness and tubular breathing disappear but reappear when the erect posture is again maintained. A valuable sign.

The Roentgen rays. Guided by my individual experience, I know of no means simpler and attended with less danger of error than the X-rays. By their aid, one is able to map out the contour

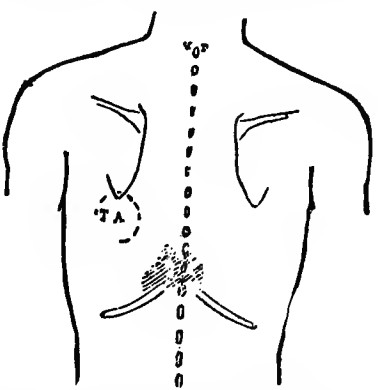


Fig. 10—The posterior pericardial patch of dullness sign (shaded) and Bamberger's sign (T A). The posterior pericardial patch of dullness is shaded. T A—Posterior patch of tubular breathing and egophony.

of the heart in its entirety. One can always detect in the normal heart some movement especially in the left ventricle. Such movements are not discernible in effusions but it may happen that an evanescent wave transmitted to the fluid by the heart may lead to an error in diagnosis. If, how-

ever, one provokes the *heart reflex,* the danger of misinterpretation is reduced to a minimum. The reflex is a phenomenon observed by means of the X-rays. It is a momentary contraction of the heart muscle upon application of an irritant to the skin of the precordia. Stroking the skin with a lead pencil or the finger nail suffices to call forth the reflex. The elicitation of the reflex is impossible in effusion.

DIAGNOSIS.

There are three characteristic signs of a pericardial effusion. 1. The apex beat located by palpation or auscultation is found an inch or two within the left border of precordial dullness. 2. The cardiac impulse is feeble and appreciated with difficulty. 3. The feeble and distant heart tones are in marked contrast with a comparatively strong radial pulse. 4. The shape of the figure of precordial dullness.

Dilatation of the heart offers the greatest drawback in differential diagnosis. The following facts are in favor of heart dilatation.

1. Previous history of valvular heart disease. 2. Absence of fever, pain and pressure symptoms. 3. The heart impulse is usually visible and wavy and the apex beat is visible and diffused. The shock of the cardiac tones may be felt 4. The area of dullness rarely assumes the triangular form, nor does it excepting in metral stenosis reach so high

or so low without visible or palpable impulse. 5. The tympanitic tone in the axillary region owing to lung compression often present in effusion is absent in heart dilatation. 6. The heart sounds are clear and sharp and there is no friction murmur.

CHARACTER OF THE FLUID EXUDATE.

In rheumatism, the exudate is usually sero-fibrinous, purulent in septic and tuberculous cases; hemorrhagic in nephritic, tuberculous and senile individuals. The only positive means of determining the nature of the fluid is by aspiration (paracentesis pericardii). This may be done with an hypodermic needle under aseptic conditions. The following points of election may be chosen, preference being given to the first: 1. Fifth left intercostal space, an inch and a half from the edge of the sternum. 2. Lower left part of the pericardial sac just within the margin of dullness. 3. Left costo-xiphoid angle. When the needle has entered the pericardial sac, suction is used. Puncture of the heart has repeatedly occurred without any special danger and only one fatal case has been reported. To avoid damage to the heart, the use of a trocar and canula has been suggested. A single aspiration with negative results is not sufficient to exclude fluid when the physical signs are strongly suggestive of its pressure.

COURSE AND TERMINATION.

The course of an effusion may be controlled by demarcating the figure of dullness by means of a nitrate of silver pencil. Sero-fibrinous effusions may reach a maximum in forty-eight hours and are often absorbed with equal rapidity. When the effusion lasts weeks, it is referred to as chronic. Sero-fibrinous effusions usually undergo absorption although pericardial adhesions remain. Cases that tend to a fatal end are marked by pressure symptoms; increasing dyspnea, cyanosis and failing circulation. Nervous symptoms are of grave import and unless they remit, death may occur within ten days. When a large effusion persists for weeks, death may result from cardiac asthenia. Etiology influences the prognosis, rheumatic pericarditis tends to recovery, whereas the tuberculous form is as a rule fatal.

TREATMENT.

The essential object is to aid absorption of the fluid. A variety of methods have been suggested: Blisters to the precordia are warmly recommended by Osler. Purges and diuretics may be tried. Iodide of potash and digitalis are employed. Depressing measures are always contra-indicated. Diaphoretic methods are used. Sodium salicylate has often a very favorable action in hastening absorption. Pilocarpin has been recommended but its use must be preceded by large dose of some

alcoholic to prevent collapse symptoms. When these methods fail or when death is imminent from cardiac pressure, indicated by increasing dyspnea, cyanosis and small rapid pulse, procrastination is fatal and recourse must be had to tapping. Puncture is usually made in the fifth interspace an inch and a half from the left sternal margin with the strictest asepsis and the amount of liquid withdrawn should not exceed 2-3 ounces at any one time. It is wiser to repeat the puncture several times rather than to remove the pressure too suddenly from the heart. If possible, the patient should be tapped in the recumbent position, for in this decubitus, the heart being heavier than the fluid sinks toward the back and is out of reach of the needle. In addition to aspiration, some writers recommend the subsequent injection of iodin dissolved with potassium iodide in water. Aspiration is generally successful if not too long delayed.

PURULENT PERICARDITIS.

This form is characterized at the onset by frequently recurring rigors, intermittent type of fever, early prostration and a rapid and unfavorable course. The etiology and symptomatology suggest the character of the fluid and aspiration proves it.

The treatment is essentially surgical. Paracentesis is not sufficient to cure it. Incision and drainage are essential and should not be delayed.

The prognosis is comparatively good after pericardiotomy for pyopericardium. Roberts collected 26 cases, showing 10 recoveries and 16 deaths. Of the fatal cases, 9 were septic, and all the others which died had severe complications.

Chronic Adhesive Pericarditis.—Adherent Pericardium.

ETIOLOGY AND PATHOLOGY.

Results from the acute form. The adhesions (synechia) may be partial or general leading to complete obliteration of the pericardial sac. The outer surface of the pericardium may become adherent to the pleura, chest wall or mediastinal tissues. The heart muscle shows atrophic and degenerative changes.

SYMPTOMS.

Inspection and Palpation. Retraction of the interspaces and even the ribs at the time of systole of the ventricles. Dislocation of the apex outward and increase of the area of impulse caused by the cardiac hypertrophy which frequently complicates the synechia. A quick rebound, known as the diastolic shock occurring after systole is regarded as characteristic. Collapse of the cervical veins (sign of Friedreich) occurs during diastole of the heart. Inspiratory swelling of the veins of the neck (sign of Kussmaul) may be observed. The pulsus paradoxus is sometimes present. It is a

pulse small and feeble during inspiration and gains strength and volume during expiration.

Percussion shows increase in cardiac dullness especially upward and to the left. When pleural adhesions complicate the trouble, the area of cardiac dullness is not diminished when the patient takes a deep breath.

Auscultation may reveal the signs of dilatation or hypertrophy.

TREATMENT.

This concerns itself with the nutrition of the heart muscle on the lines indicated in the treatment of valvular lesions. The embarrassed heart may stop suddenly in fatal syncope or pass through the stages of broken compensation.

MEDIASTINO-PERICARDITIS.

ETIOLOGY AND PATHOLOGY.

Occurs most frequently in young adults and males from an extension of the pericardial inflammation to the anterior mediastinum. The pericardium is thickened and adherent to the structures in the anterior mediastinum.

SYMPTOMS.

Dyspnea, cyanosis, venous engorgement, liver enlargement, ascites and anasarca. The physical signs are those of adherent pericardium. The *mediastinal friction,* systolic in time, heard over

the sternum and increased in intensity when the arm is raised has been observed by Perez.

Hydropericardium.—Dropsy of the Pericardium.

ETIOLOGY AND PATHOLOGY.

The occurrence of fluid in the pericardium without inflammation of the serous sac. The serous transudate is secondary and associated with cardiac or renal dropsy when other serous cavities are similarly occupied by fluid. Fluid may accumulate suddenly in nephritis especially in the scarlatinal form. Intra-thoracic mechanical causes may contribute to the accumulation of a non-inflammatory fluid in the pericardium. When the serum has a milky character it is known as chylo-pericardium.

The symptoms are those of effusion without fever or friction murmurs. The treatment is that indicated in general dropsy although aspiration may be necessary.

HEMOPERICARDIUM.

The causes are: Rupture of the first part of the aorta, the coronary arteries or the heart. Wounds of the heart and pericardium are further causes. Death may occur before symptoms develop especially in ruptured aneurisms. In tuberculosis and cancer, the effusion may be blood-stained and must not be regarded as instances of hemopericardium. Death results from heart fail-

ure, the result of compression. Aspiration has been successful in a limited number of traumatic cases.

PNEUMOPERICARDIUM.

Air or gas in the pericardial sac is rare and is caused generally by perforated thoracic wounds or the result of perforation from the lungs, stomach or esophagus. Decomposition of pus in the sac may develop gases. When pus is present, we speak of a pyo-pneumopericardium. The physical signs are those yielded by the pressure of fluid and gas. Percussion gives a movable arc of dullness by altering the patient's posture with a tympanitic sound in the region of the gas. The heart sounds on auscultation assume a metallic splashing character. Death rapidly occurs unless the trouble is caused by perforation from without.

Treatment is indicated in the latter instance by enlargement of the wound and free incision. Air is sometimes spontaneously absorbed as in pneumothorax.

CHAPTER V.

ENDOCARDITIS AND CHRONIC VALVULAR DISEASE.

Endocarditis.

Etiology and Pathology.

Inflammation of the lining membrane of the heart is usually confined to the valves and is generally a secondary infection in the course of various diseases. The pathologic antecedent is generally acute articular rheumatism, the etiologic elements of which have not yet been established. The arthritic phenomena may be secondary to the endocardial inflammation. When secondary to erysipelas, the streptococcus pyogenes may be demonstrated. In the suppurative processes, like pyemia and puerperal fever strepto and staphylococci are found. Endocarditis following croupous pneumonia and pulmonary tuberculosis is not uncommon. Osler in 100 autopsies in pneumonia cases found it present in 5 instances and in 216 necropsies on phthisical cases, it was present in 12 instances. Diphtheric endocarditis is not frequent and the same statement applies to typhoid endocarditis which is caused by the typhoid bacillus.

In gonorrheal endocarditis which is not infrequent, the gonococcus has been frequently demon-

strated in the endocarditic vegetations. In the endocardial inflammation complicating acute nephritis, the micro-organisms concerned in producing the nephritis are the exciting agents.

Pathologically the different forms of endocarditis are characterized as follows: Simple acute endocarditis shows the presence of minute vegetations on the valves of a warty appearance. These vegetations may be absorbed, result in the production of an ulcer or end in chronic valvulitis with deformity.

Malignant or ulcerative endocarditis is characterized by rapidly occurring ulceration of the valves, heart septum or the heart itself. Suppuration may complicate the ulceration.

Chronic Endocarditis is an interstitial inflammation of the heart valves leading to deformity of the valve segments. It is a slow process and is the usual cause of chronic valvular disease. Syphilis, gout, alcoholism and prolonged muscular exertion are the usual causes.

DIAGNOSIS.

Simple Endocarditis. The subjective symptoms are usually negative. The physical signs are alone conclusive. In the course of an infectious disease, cardiac complication is betrayed by palpitation and irregularity of the heart.

The physical signs are evident by auscultation. Murmurs or roughened heart sounds may be pres-

ent. Very frequently the physical signs are dubious. The occurrence of fever of moderate range (100-102 deg. F.) together with a murmur over one of the heart orifices with perhaps irregularity in the organ speak for endocarditis. One must not mistake the soft bellows murmur often heard in acute febrile diseases usually heard over the aortic area with the murmurs occurring in endocarditis which are best heard over the mitral area.

Malignant endocarditis presents two distinct types, the septic or pyemic and the typhoid. The septic type associated with wounds and septic processes is characterized by chills, sweats, irregular fever and the usual phenomena of septic infection. This type has been known to be frequently mistaken for intermittent fever.

The typhoid type is more frequent than the former and is manifested by irregular temperature, delirium, prostration, coma, diarrhea and sweating. Petechial rashes and erythema are common in both types as well as embolic phenomena. The emboli take their origin from the soft vegetations on the valves and are carried to the different organs. When the emboli go to the brain, delirium, coma, aphasia or hemiplegia results; to the kidney, hematuria; to the spleen, local peritonitis; to the skin, minute hemorrhages.

The physical signs are notoriously uncertain. A murmur may or may not be present. A murmur

varying in character from day to day is characteristic of malignant endocarditis. Malignant endocarditis may develop in consequence of infection on an old valvular heart lesion. The diagnosis is easy when embolic phenomena occur associated with irregular fever, profound prostration and the presence of heart symptoms.

DIFFERENTIAL DIAGNOSIS.

From malaria, endocarditis of a malignant type may be excluded by an examination of the blood. From cerebro-spinal fever, we must rely on the preponderance of cardiac symptoms. From typhoid fever, with which disease it is most frequently confounded, the following symptoms speak against typhoid fever and for malignant endocarditis; history of rheumatism, pneumonia or some infectious disease, no prodromata, onset marked by a severe chill, rapid rise of temperature of an irregular type, profound prostration early, embolic symptoms (hemiplegia, aphasia, hematuria, etc.), cardiac symptoms (loud systolic murmur), septic leucocytosis.

Chronic Endocarditis manifests itself by the presence of symptoms peculiar to chronic valvular disease which will be considered under special lesions of the valves.

COURSE AND TERMINATION.

In simple acute endocarditis, there is rarely any immediate danger, the prognosis depending on the

character of the primary disease. As a rule, this form of endocarditis is the initial factor in the development of permanent valvular lesions of the heart. In malignant endocarditis the prognosis is likewise dependent on the primary disease. Unless grafted upon a chronic valve lesion, the disease rapidly tends toward a fatal termination, the course rarely lasting more than six weeks, whereas in some instances, the disease may terminate fatally in a few days. In one of my patients with gonorrheal endocarditis, the disease lasted only three days. It was marked by emboli which completely cut off the circulation in three of the fingers of one hand. In the chronic form, the prognosis is that of the individual lesions of the valves.

TREATMENT.

No measures are yet known by which endocarditis can be prevented although absolute rest in bed and protection of the body against cold in the specific fevers, may diminish the tendency to the disease. The value of the salicylates in rheumatism while undoubted have little influence in preventing endocarditis. We have no remedy which will directly influence the endocarditis, although something may be done in the way of symptomatic treatment. *Rest* must be enjoined in all cases and vascular excitement controlled by the ice bag to the precordia and the use of aconite. *Heart failure* calls for strychnin and alcoholic stimulants,

while digitalis is positively contra-indicated, the drug causing violent cardiac contractions of an inflamed and enfeebled heart. In the malignant form of endocarditis, antistreptococcus serum promises to be of some value.

Chronic Valvular Disease.

aortic incompetency; aortic insufficiency; aortic regurgitation.

General Symptoms. If perfect compensation exists, there may be no symptoms. *Arterial anemia,* especially of the brain, is an early symptom and the patient complains of attacks of giddiness, is pale and suffers from dyspnea. Pains in the region of the precordia and radiating to the neck and arms occur more often in this, than in any other valvular lesion of the heart.

Physical Signs. They are made up of the evidence furnished by hypertrophy of the left ventricle, viz., dislocation of the heart apex, downward outward and to the left, increased area of cardiac impulse, increased area of cardiac dullness, which is greater than in any other valve lesion, and which is increased downward and to the left. The chief sign of this lesion is obtained by *auscultation;* at the second right costal cartilage a diastolic murmur is heard.

characteristics of the murmur of aortic incompetency.

1. It is propagated along the sternum toward

the apex. 2. Its point of maximum intensity may be the fourth left costal cartilage on the apex. 3. It may be heard in the vessels of the neck. 4. The murmur is usually soft, but sometimes rough and loud. 5. A systolic murmur heard in the aortic area is not diagnostic of aortic stenosis, it is more often caused by roughening of the semilunar valves or of the inner coating (intima) of the aorta. 6. A systolic murmur heard in the mitral area, associated with aortic regurgitation may be caused by relative insufficiency of the mitral opening.

Arterial Signs. The peripheral vessels pulsate more often in this than in any other valve lesion. Double murmurs may be heard over the carotids and subclavians. The *water hammer or Corrigan pulse* is characteristic, a quick and jerking pulse which, striking the finger, rapidly recedes. This pulse phenomenon is accentuated when the arm is elevated. The *capillary pulse* is obtained by drawing a line with the finger nail across the forehead. The hyperemia induced on either side of the line, becomes alternately red and pale. It is also seen beneath the finger nails.

Course and Termination. The lesion may be compensated for years without inconvenience. The occurrence of heart degeneration marks the advent of disturbed compensation, beginning with precordial pain, headache, vertigo, palpitation,

cardiac distress, edema and dyspnea. General dropsy is not common unless a mitral lesion complicates the trouble. *Sudden death* is more frequent in this, than in other lesions. With compensation failure, slight irregular fever and embolic phenomena due to *recurring endocarditis* terminate the scene.

AORTIC STENOSIS

General Symptoms. Owing to narrowing of the aortic orifice the deficient systemic blood supply induces most frequently signs of *cerebral anemia.*

The physical signs are those common to left ventricular hypertrophy. Palpation may detect a systolic thrill in the aortic area. Auscultation reveals a murmur in the aortic area, systolic in time and transmitted along the course of the blood vessels. The murmur is harsh, loud and sometimes musical. The second sound, if regurgitation is not present may be muffled or absent. This is caused by stiffness or thickening of the valve.

Diagnosis. A systolic murmur in the aortic area may also be caused by atheroma or dilatation of the aorta, or anemia. A murmur due to the first causes is often accompanied by a second sound which is accentuated and the small and slow pulse and systolic thrill are absent. The murmur of anemia is also accompanied by an accentuated sec-

ond tone and there is no hypertrophy, thrill, or small pulse. Signs of anemia are present.

Course. If hypertrophy is present, the condition may be latent. The early signs of compensation failure are: Dizziness, pain in the precordia and palpitation.

MITRAL INCOMPETENCY; MITRAL REGURGITATION; MITRAL INSUFFICIENCY.

General Symptoms. The effects of this lesion on the pulmonic and systemic circulation after failure of compensation is more pronounced than disease at any of the other orifices. As in other lesions, there are no symptoms if the trouble is compensated. When compensation fails, we have all the characteristic symptoms of heart disease, cyanosis, dyspenea, cough and expectoration, dropsies, etc.

The physical signs are those of dilatation and hypertrophy of both chambers at the time of full compensation. *Auscultation* exists in the mitral area, a murmur *systolic* in time, transmitted to the left axilla and scapular angle. In accordance with hypertrophy of the right ventricle and consequent increased tension in the pulmonary artery, we hear accentuation of the second pulmonic tone.

Diagnosis. The systolic murmur of *aortic stenosis* and tricuspid regurgitation may be mistaken for mitral incompetency. The following

data speaks for *aortic stenosis:* The murmur is loudest over the base and is transmitted to the vessels of the neck, there is no accentuation of the second pulmonic tone, the left ventricle only is enlarged, the thrill if palpable is at the base of the heart. In tricuspid regurgitation, we have pulsation of the cervical veins, pulsation of the liver, and the systolic murmur has its seat of maximum intensity at the base of the ensiform cartilage; the propagation of the murmur is not so extensive nor in the direction of the murmur of mitral incompetency. We must also exercise care in distinguishing functional murmurs from the murmur of incompetency.

MITRAL STENOSIS.

General Symptoms. Constriction of the left auriculo-ventricular orifice may exist for years without symptoms, although at any time a fresh endocarditis may develop and give rise to the phenomena of embolism in the brain or viscera.

The physical signs are pathognomonic of this lesion provided compensation exists. The brunt of the burden is borne by the right auricle and ventricle which become hypertrophied. The left ventricle does not participate in the cardiac changes.

Inspection. In children, rarely in adults, the hypertrophied right ventricle manifests its presence by bulging of the lower sternum and fifth and

sixth left costal cartilages. The apex beat is only slightly displaced.

Palpation. As a rule a pronounced fremitus or thrill is felt in the fourth or fifth interspace within the nipple line. This thrill is characteristic and may be the only reliable sign of the lesion. The thrill is rough and grating, limited in area and culminates in a sharp sudden shock. The cardiac impulse is felt in the third and fourth interspaces and is due to an enlarged right ventricle.

Percussion yields increased dullness to the right of the sternum and increased dullness upward as high as the second rib.

Auscultation. In the mitral area, usually limited, a murmur of a churning and grinding character is heard which is synchronous with the thrill and terminates with a loud shock that is heard at the same time as the first sound. Like the thrill, this murmur is pathognomonic. This murmur is the presystolic murmur, occuping the entire period of diastole or more often the latter half of this phase. The second pulmonic sound is accentuated. The pulse is smaller in volume than normal, but regular.

There are associated murmurs, chief of which is the mitral systolic, as stenosis rarely occurs without some incompetence of the valve. A tricuspid systolic murmur may be present owing to relative insufficiency of that valve.

TRICUSPID INCOMPETENCY; TRICUSPID REGURGITATION.

This rarely occurs as a result of valvular endocarditis. As a rule, it is a relative insufficiency superinduced by dilatation of the right ventricle, secondary to lesions of the valves on the left side or pulmonary diseases, causing obstruction to the circulation.

The symptoms are mainly revealed by physical signs and are made up of phenomena associated with obstruction in the pulmonary circulation and systemic veins.

Diagnosis. 1. Pulsation of the veins of the necks, caused by systolic regurgitation of blood into the right auricle and the transmission of the pulse wave into the cervical veins. The right jugular vein pulsates more forcibly than the left. Regurgitation into the vein is associated with the pulsation. To observe the phenomenon of regurgitation, empty the external vein by pressing on the same just above the clavicle and moving it along the vein in the direction of the lower jaw. Thus emptied, with each cardiac systole, it will be observed to fill up from below.

Regurgitant pulsation may be transmitted to the inferior vena cava and thence to the hepatic veins, causing hepatic venous pulsation. Hepatic pulsation is best felt by bimanual palpation, one hand over the fifth and sixth costal cartilages and the

other over the liver in the axillary region. 2. A systolic murmur in the tricuspid area. It is usually soft, and blowing and may be absent. Percussion shows increased cardiac dullness to the right of the sternum.

TRICUSPID STENOSIS.

Very rare and usually fatal in origin. Other congenital lesions may mask its presence. The physical signs are those of mitral stenosis, with transference of the signs to the right side. Extreme cyanosis is common and dropsy extreme. A positive diagnosis is rarely made owing to its association with other lesions.

PULMONARY VALVE LESIONS.

Stenosis is one of the rarest of acquired lesions, but the most frequent of the congenital heart affections. The congenital lesion is associated usually with patency of the ductus Botalii and defect of the ventricular septum. Cyanosis and dyspnea are extreme. Auscultation shows a systolic murmur in the second left interspace.

Insufficiency. Like the foregoing it is usually congenital, but may arise from endocarditis or be merely relative from dilatation of the pulmonary artery at its origin. The murmur replaces the second pulmonic sound, and its intensity is increased during expiration. There is hypertrophy and dilatation of the right ventricle.

COMBINED VALVULAR LESIONS.

In more than one-half of all the cases of cardiac valvular lesions, combined murmurs are present. Stenosis of a valve is, as a rule, combined with insufficiency of the same valve. Thus aortic stenosis and insufficiency coexist, but one may for a time compensate the other so that only the evidence of one lesion is demonstrable. Such a lesion as the one just cited would act as follows: The stenosis diminishes the regurgitated quantity of blood from the aorta into the left ventricle. Relative insufficiency of the mitral valve sequential to aortic insufficiency counteracts overfilling of the left ventricle and also over-distension of the aorta.

A relative tricuspid insufficiency secondary to mitral disease may be doubly interpreted. Such a lesion may be speedily fatal owing to over-distension of the general venous circulation, or it may prove salutary because it may relieve the right ventricle of its surplus of blood. The combined valvular lesions in order of frequency are: 1. Mitral and aortic segments. 2. Mitral and tricuspid lesions. 3. Aortic, mitral and tricuspid.

Aortic insufficiency or aortic stenosis exists more frequently in combination with mitral insufficiency than aortic stenosis with mitral stenosis or mitral stenosis with aortic insufficiency. The most frequent association in adults is mitral insufficiency with slight aortic stenosis, whereas in children the

most common association is aortic and mitral insufficiency.

DIAGNOSIS.

Valvular lesions are not difficult of location, even though several murmurs coexist, provided compensation is present. The average duration of compensation, based on a study of 102 cases by Romberg, has been found to be seven years. Sooner or later compensation fails and the heart becomes rapid and irregular with faint sounds and murmurs, a condition spoken of as *delirium cordis*. When this heart delirium occurs it is almost impossible to correctly time the murmurs. Regulation of the cardiac action with digitalis and physical rest may prove of advantage, but until some regulation is established it is often impossible to make a correct diagnosis.

Differentiation of contemporaneous murmurs may be possible by percussion, auscultation and the inhibition maneuver. The secondary changes in the myocardium usually coincides with the predominating murmur. If auscultation determines two murmurs of different character, one blowing and the other rough, two distinct murmurs exist. If again, we hear, let us say, a murmur at the apex and another at the aorta, auscultate step by step from one situation to the other. If it is everywhere audible, but becomes louder toward one

point, then its origin is at the latter situation and is conveyed to the other.

The inhibition maneuver described in the chapter on Diagnosis is an invaluable aid in causing transmitted murmurs to disappear to be replaced by tones. *The maneuver should only be attempted after forced expiration,* for when the lungs are inflated all endocardial murmurs are naturally weakened.

CONGENITAL HEART DISEASE.

The most frequent lesion is stenosis of the pulmonary orifice, associated very often with imperfections of the ventricular septum and patency of the foramen ovale and ductus arteriosus. In 86 per cent of patients with congenital heart disease living beyond the twelfth year, according to Peacock, the lesion is at the pulmonary orifice.

Symptoms. Cyanosis is the chief symptom in over ninety per cent of the cases, hence the terms "blue disease" and "morbus ceruleus," which are other names for congenital heart disease. The lividity appears in the first week of life. The skin may be universally purple or may be confined to the fingers, lips, nose and ears. It is increased by exertion. Dyspnea and cough are common. Physical development is retarded and the mind is sluggish. Clubbing of the fingers and toes is a common occurrence.

Diagnosis. Cyanosis in children with or with-

out enlargement of the heart, together with a murmur during the early weeks of life, is due to congenital heart disease.

Prognosis. More than one-half the patients die before the end of the first year, and not less than three-fourths before the end of the third year.

MYOCARDITIS.

Inflammation of the myocardium may be acute or chronic. Etiology. The acute specific fevers due to the infectious element. The chronic variety is associated with atheroma, and frequently complicates chronic Bright's disease.

The acute form may result in dilatation of the heart, fatty heart or aneurism of the heart. The chronic form may result similarly.

Symptoms. The diagnosis, myocarditis is made more often by the pathologist than the clinician for the symptomatology of the disease is vague and uncertain. If in the course of an acute specific fever, precordial oppression, dyspnea and syncope occur and if to these symptoms, we add a rapid and weak pulse, signs of cardiac enfeeblement and the physical signs pertinent thereto, we may suspect myocarditis.

The recognition of cardiac aneurism is made possible by careful percussion of the heart. The latter sign shows a projection beyond the line of cardiac dullness. With the Roentgen rays, I was able in one patient to trace with accuracy the ir-

regular outline of the heart and the diagnosis was confirmed at the necropsy, death having occurred suddenly after exertion from rupture of the heart, a frequent sequel in cardiac aneurysm.

Treatment. Absolute physical rest and proper feeding are indicated. No drug beyond the use of strychnin is of advantage. Iodide of potash long continued is said to promote the nutrition of the heart. The Nauheim system of baths and resisted movements have given me marvelous results in a few cases. In some instances the movements have been harmful. It is difficult to define indications for the baths and movements, the contraindications are evidenced by the results of such treatment.

FATTY HEART.

Two pathologically distinct affections must be differentiated: 1, fatty degeneration in which the muscle fibers of the heart have been transformed into fat, and 2, fatty overgrowth in which the normal epicardial fat is increased in amount.

FATTY DEGENERATION.

Etiology. Nutritional disturbances of old age and the wasting diseases. Infectious fevers, chronic anemia, arsenical and phosphorus poisoning, diseases of the coronary arteries and finally as a secondary lesion in cardiac hypertrophy.

Symptoms. Diagnosis is, as a rule, obscure. The chief sign is cardiac enfeeblement. Cardiac

asthma, angina pectoris, pseudo-apoplectic attacks and pulse retardation (30-40 beats per minute) are relatively frequent. Cheyne-Stokes breathing and the fatty arcus senilis, formerly regarded as pathognomonic, are untrustworthy.

FATTY OVERGROWTH.

An increase of subpericardial fat is usually a manifestation of general obesity. The recognition of the condition is based on the general obesity associated with signs of heart failure, viz.: Asthma, syncopal attacks, bronchitis with weak and muffled heart sounds. Sudden death occurs from syncope or from rupture of the heart.

Treatment. The treatment of fatty degeneration is strictly symptomatic. Fatty overgrowth is greatly benefited by the method of Oertel referred to under general treatment as well as by the Schott method.

CHAPTER VI.

NEUROSES OF THE HEART.

The rapidity and force of cardiac action are regulated by the pneumogastric or vagus nerve which inhibits it and the sympathetic which accelerates it. In the heart the blood pressure is regulated by a branch of the vagus, the depressor nerve, which acts by causing sudden dilatation of the large abdominal vessels to lessen cardiac pressure or by constricting them to raise it. The vaso motor system of nerves regulates the caliber and tone of the blood vessels. It is connected with the heart, so that tension of the arteries and force of the cardiac pulsations are regulated with each other.

The coronary arteries are the nutrient vessels of the heart. They arise from the aorta immediately behind the valve and their blood is returned by a vein to the right auricle, where its opening is guarded by a little valve.

GENERAL ETIOLOGY.

Largely reflex from the stomach and intestines. Peripheral irritation of the gastric branches of the vagus by the products of indigestion is a fruitful cause. How this irritation is induced is as yet

a conjectural matter, although we do know that when treatment is directed toward the relief of a gastric affection cure is often attained.

The absorption of substances from the intestinal tract which are the result of bacterial activity, must also be taken into consideration, and while we possess no means of demonstrating such products in the circulation we assume that they exist owing to the good results following treatment. Cleansing the intestinal tract is often a herculean undertaking, but like the fabled stables of Augeas our endeavors must be more in the direction of asepsis than antisepsis.

The genito-urinary apparatus of both sexes is frequently implicated in the etiology of cardiac neuroses and demand careful investigation. A similar statement is apposite with reference to the naso-pharynx. Anemia is a common cause and so is the inordinate use of alcohol, tea, coffee and tobacco. Mental excitement, depression or emotion is a causative factor.

In a number of individuals no etiologic factor beyond a neurasthenic condition may be demonstrated, and it would appear in these cases as if the cardiac apparatus bore the brunt of the insanity of the nervous system representing, as it were, the *locus minoris resistentiae*. At any rate I have known the most intractable cardiac neuroses yield to a thorough rest cure.

I. Palpitation.

This term is applied to conscious cardiac contractions of the heart of increased force associated with a disturbance of rhythm and sometimes with distress in the precordia, dyspnea and anxiety. Besides the factors previously mentioned in the general etiology, the nervous phenomenon may be associated with organic heart disease, although this is infrequent. The irritable heart described by Da Costa, common among the young soldiers during the Civil war, is a similar neurosis. Two facts were concerned in its causation, mental excitement and excessive muscular exertion.

DIAGNOSIS.

Visible cardiac pulsations against the chest wall, pulse 120-160 per minute and loud cardiac tones are practically the objective symptoms of a paroxysm which may last from a minute to an entire day. A mild paroxysm, often the result of indigestion, is attended by a slight fluttering of the heart and a sensation which the patient describes as a "goneness." The diagnosis of nervous palpitation should only be made when careful examination of the heart reveals no evidence of organic disease. A murmur must not be construed as evidence, insomuch as it is often hemic, and anemia is largely concerned in the causation of the neurosis.

TREATMENT.

Suggestion plays an important rôle. Convince the patient that the trouble is purely functional and half the battle is won. To logically carry out this suggestion medicines are contra-indicated; as much may be effected by hygienic measures. Regulating the methods of living, careful dieting, avoidance of alcohol, coffee, tea and tobacco, interdicting sexual excitement and mental excitement, bowel regulation and a modified rest cure are a few hygienic regulations.

The paroxysm of palpitation may be arrested by certain mechanic manipulations, especially in hysterical persons, by pressure on the vagus in the neck and certain hysterogenic zones on the abdomen, particularly the ovarian region. Rest in bed and an ice bladder to the precordia may also be tried. The bromides, valerian, camphor and hyoscyamus may prove beneficial, but the most effective remedy is unquestionably morphin when given hypodermically. Recurrent paroxysms may be prevented by observing indications for therapeutic measures, the treatment of anemia, hysteria, malaria, gout and the uric acid diathesis. Galvanism of the vagus is sometimes beneficial. The continued use of tincture of nux vomica in large doses is particularly valuable. One of my patients, a physician, suffering from palpitation for ten years, found almost immediate and permanent relief

from the Schott methods of resistance exercises and baths.

II. Paroxysmal Tachycardia (Rapid Heart).

This is a paroxysmal affection variable in duration, associated with a feeling of great anxiety, in which the number of pulse beats may reach 150 or more. Two forms have been described, *neurotic* and *symptomatic tachycardia.* The causes of the former variety are the same as in palpitation. The symptomatic variety may be due to central and peripheral causes.

Central causes: lesions of the brain and cord.

Peripheral causes: tumors, aneurisms, enlarged lymph glands which compress the vagus and neuritis of the vagus.

The rapid heart is directly dependent upon either paralysis of the vagus or stimulation of the sympathetic nerves. Fraentzel suggested that the cause could be ascertained by digitalis and morphin. If the vagus were at fault the former drug would prove effective, whereas if the sympathetic were at fault morphin would prove useful.

DIAGNOSIS.

Heart hurry is characterized by paroxysms of a high pulse rate (in one of my patients 300 beats per minute) without a palpable cause, dissociated with any cardiac anomaly in the inter-paroxysmal periods. Nothnagel decides that a great increase

in the pulse frequency, accompanied by a weak heart beat, speaks for paralysis of the vagus, whereas a strong impulse, fullness of the peripheral arteries with high tension is in favor of stimulation of the accelerators. This condition must not be confounded with a normally rapid pulse nor with an increased pulse rate occurring in certain pathologic conditions.

TREATMENT.

The same general methods recommended in the treatment of palpitation are here applicable. Digitalis has been serviceable, but no dependence can be placed on its action. Subjugation of the paroxysm of tachycardia may be accomplished by galvanization of the vagus (positive pole under angle of jaw, negative pole lower down over each side of neck). In a case reported by Nothnagel, attacks were jugulated by deep inspirations. Rosenfeld's patient controlled her attack by going to bed, raising her head with her feet planted firmly against the foot of the couch, and then taking a forced inspiration she pressed down with all her might, with the object of closing the glottis. Schott warmly recommends his balneologic and gymnastic methods. The long-continued use of iodide of potash proved curative in one of my patients. A colleague controlled his attacks with digitalis. He had tried twelve preparations of

the tincture from as many different drug stores without any result. A thirteenth preparation from an homeopathic pharmacy was succcessful.

III. Brachycardia (Bradycardia—Slow Heart).

Slowness of the pulse may be physiologic. Napoleon had a pulse of only 40 per minute. Before deciding whether brachycardia really exists it is necessary to determine if the arterial and heart-beats correspond, for while the cardiac pulsations may be 70 only 30 beats reach the radial pulse, therefore the cardiac contractions and not the pulse beats should be counted. Riegel's classification of brachycardia is the one usually accepted.

Physiologic brachycardia.—In the puerperal state a slow pulse is a common manifestation when it may reach a rate as low as 34.

Pathologic brachycardia is present in convalescence from acute fevers, notably rheumatism, diphtheria, pneumonia and typhoid fever. The cause is most probably resident in the heart muscle and not dependent on exhaustion as maintained by Traube.

Diseases of the digestive organs was the chief etiologic factor in Riegel's cases. Diseases of the lungs.—In valvular heart lesions it is not common, although in degeneration of the heart muscle it is frequent. Cases of fatty heart have been ob-

served where the pulse rate was only 12 per minute, and this rate was maintained for years. Nephritis, toxic agents, diabetes, anemia, diseases of the cord and brain are regarded as other causes. Brachycardia arising reflexly from some disturbance in the gastro-enteric tract is easily understood when we remember how readily the inhibitory action of the vagus may be excited through this channel. In diseases of the heart, brain and kidneys it is often an ominous sign. It is often a symptom in uremia. Muscarin and the biliary salts can produce a slow pulse. Rapid resorption of large quantities of bile not only slows the pulse but makes the heart action irregular. Thus, in catarrhal icterus a slow pulse is a common occurrence.

SYMPTOMS.

During a paroxysm. Syncopal attacks occur and the patient may remain unconscious for hours. During the attack the heart impulse and sounds are feeble. Sudden death may terminate an attack.

TREATMENT.

Rest is essential. The treatment is mainly symptomatic, although a thorough examination may often determine a causal condition, the removal of which cures the affection. To excite the action of the heart in a paroxysm, caffein, strychnin and nitro-glycerin may successively be tried.

IV. Arrhythmia (Irregular Heart).

An irregular heart may be clinically manifested as an *intermission* when one or more beats of the heart are dropped, or as an *irregularity* when the beats show inequality in volume and force. Arrythmical action is expressed by the following well recognized varieties of pulse:

1. The paradoxical pulse, in which during inspiration the beats are more rapid though less full than in expiration. It attends chronic adhesive pericarditis when fibrous bands become attached to the root of the aorta. It may be felt in the sleeping child.

2. Intermittent pulse signifies a missed or dropped beat. This intermittency may be irregular or cyclic, an intermittence occurring at every fourth, sixth or eighth beat.

3. The alternate pulse is expressed by alternate full and feeble pulse beats.

4. The bigeminal pulse occurs when two beats follow each other quickly and the next two not so quickly, three such beats occurring in rapid succession gives rise to the trigeminal pulse.

5. The pulse of delirium cordis gives rise to marked irregularity and inequality of the pulse beats.

Irregularity of heart rhythm may give no expression in the pulse. We have embryocardia or fetal heart rhythm in which shortening of the

long pause exists, and the first and second sounds as in the fetal heart are similar. This sign is of ominous import in fevers, indicating a weak heart. Gallop or cantering rhythm, expressed by the words "rat ta-tat," are sounds simulating the triple foot-fall of a horse at canter. Present in arterio-sclerosis, interstitial nephritis and myocarditis. It may be met with in health.

ETIOLOGY.

The causal classification of Baumgarten is usually accepted: 1. Organic cerebral affections. 2. Reflex from diseases of the viscera. 3. Toxic; tobacco, coffee, tea and from such drugs as digitalis, belladonna and aconite. 4. Changes in the heart.

SIGNIFICANCE.

Arrhythmia may exist for a long period without symptoms. It is usually in association with other cardiac signs that its presence is noted. Associated with myocardial or valvular lesions it is ominous, but as a permanent condition secondary to mental influences it is usually without significance. The treatment is symptomatic.

ANGINA PECTORIS (STENOCARDIA—BREAST PANG, CARDIODYNIA).

A symptomatic paroxysmal affection (described by Heberden as the breast pang) associated with cardiac lesions.

ETIOLOGY AND PATHOLOGY.

An affection of adult life occurring chiefly in men. Associated, as a rule, with arterio-sclerosis, hypertrophy of the heart and lesions of the myocardium and aorta. No hypotheses yet advanced suffice to account for its symptomatology. The hypotheses thus far advanced are: 1. That it is a neuralgia of the cardiac nerves. 2. A cramp of the heart muscle (Heberden). 3. Extreme tension of the ventricular walls following acute dilatation with involvement of the coronary arteries (Traube). 4. Spasm of the coronary arteries with increased intra-cardiac pressure. In fatal cases the coronary arteries are usually diseased. In one of my patients the coronary arteries were practically calcareous tubes, yet the pulse showed no evidence of arterio-sclerosis with the sphygmograph.

SYMPTOMS.

The paroxysm begins suddenly, usually after some exciting cause. There is agonizing pain in the heart region, radiating up the neck and down the arms, particularly to the left arm. The sensation is one of impending death and the feeling one as if the heart were held in a vise. The face is pale and bathed in perspiration. Dyspnea is not the rule. Little or no changes are noted in the pulse or heart during an attack. The paroxysm is of short duration (few seconds to three min-

utes) and is followed by eructations of gas, vomiting or discharge of a large quantity of clear urine. The attacks may recur at intervals of from weeks to years. The chief diagnostic points are: 1. Sudden intense pain and sense of impending death. 2. Occurrence in men between the ages of 40 and 60. 3. Existence of arterio-sclerosis characterized by accentuated second aortic tones and pulse of high tension. I can recall two individuals who for years suffered from slight pains in the left arm with numbness in the hand and fingers who eventually died in a typical attack of angina.

A variety of the true form of angina has been described by Nothnagel as angina vasomotoria. This form follows exposure to cold and is characterized by a general spasm of the peripheral arteries with pallor of the face and coldness and stiffness of the limbs. The chief difficulty in diagnosis is to differentiate the true from the *false* or *hysterical pseudo-angina.* The chief diagnostic signs of pseudo-angina are: 1. Occurrence in hysterical women and neurasthenic men. 2. Occurrence at every age. 3. Attacks are periodical, spontaneous and often nocturnal and associated with nervous symptoms. 4. Attack lasts from a half to several hours, and is never fatal. 5. Associated with extreme restlessness and emotional symptoms.

I am surprised to find in the literature very little reference to a dilated stomach as the cause of pseudo-angina, a form described by myself as *gastrectatic pseudo-angina.* I have frequently encountered this affection and permanent cures have followed treatment directed toward the stomach by

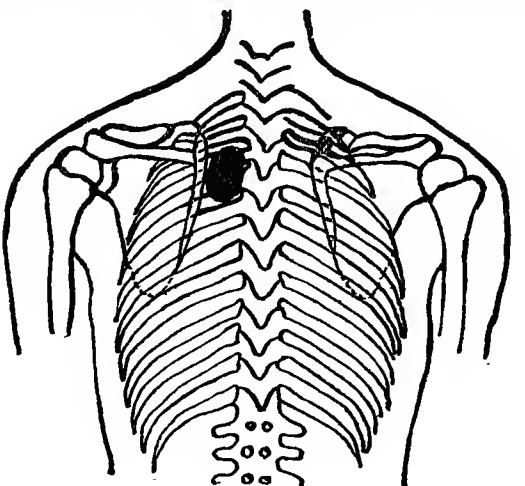


Fig. 11—Dull area in dislocation of heart upward by a dilated stomach.

a suitable dietary and lavage. If the gastric trouble is provoked by neurasthenia, the latter condition demands treatment.

In a previous chapter I have demonstrated the facility with which a dilated stomach may dislo-

cate the heart, since which time I have discovered a new and trustworthy sign of heart dislocation consecutive to gastrectasis. It is a circumscribed area of dullness, often amounting to flatness in the left interscapular region between the internal border of the scapula and spine. Over the dullness

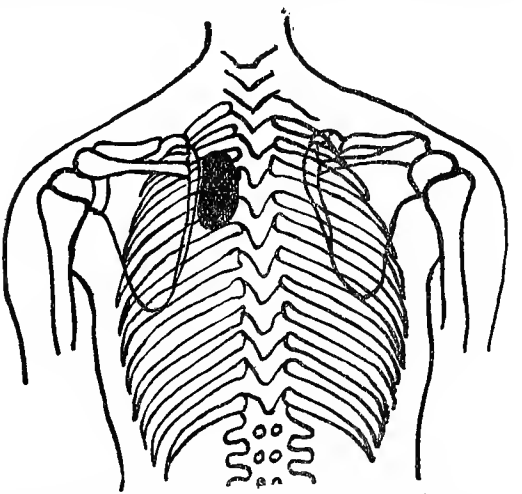


Fig. 12—Same case. Area of dullness increased by patient leaning backward.

bronchial respiration is heard. When the patient is directed to lean forward the dullness disappears and likewise the bronchial breathing, but are again in evidence when the erect attitude is resumed. When the patient is directed to lean backward the area of dullness is very much increased.

This phenomenon is caused by a dislocated heart compressing the lung, which fact is easily demonstrated by examination with the Roentgen rays. The foregoing syndrome I have reproduced synthetically by distending the stomach with air, thus proving the correctness of my conclusions. Identical percussional phenomena may be observed when the heart is enormously enlarged upwards.

The prognosis is always bad in true angina, although years may elapse before a fatal termination, provided excitement, muscular exertion and dietetic errors are avoided. Vasomotor angina is less grave and pseudo-angina is always favorable. Cardiac pain without evidence of arterio-sclerosis, or valve disease, is not of much moment (Osler).

TREATMENT.

A quiet life should be encouraged. Attacks may be curtailed and prevented by inhalations of *nitrite of amyl,* perles of which containing 3 to 5 drops should be constantly carried by individuals thus afflicted. If the attack is not controlled one minim of the 1 per cent solution of *nitro glycerin* should be given hypodermically and repeated every 15 minutes if pain continues, or until the physiologic effects (flushing of the face and headache) are evident. When this fails chloroform by inhalation or a hypodermic of morphia may be given. In the intervals between the attacks the prolonged use of the iodide of potash in 20-grain doses three

times a day may control the frequency of the attacks by influencing the associated arterio-sclerosis, especially if there is a history of syphilis.

Habitual exaltation of arterial tension is influenced by increasing doses of nitro-glycerin until a dose large enough to produce its physiologic action is attained. Sodium nitrite (dose gr. i-iii) has a similar action. In pseudo-angina the causal condition must be eliminated. Static electricity has a marked psychic action in cases of pseudo-angina. The Schott system of baths and exercises improve the condition of the heart, muscle and arteries and should be employed in the true forms of angina. Erythrol-tetranitrate in grain doses, four times in the 24 hours, is a new remedy for the relief of the anginal attacks.

CHAPTER VII.

AFFECTIONS OF THE ARTERIES.

Arterial Sclerosis — Arterio-Sclerosis; Arterio-capillary Fibrosis; Atheroma.

PATHOLOGY AND ETIOLOGY.

The normal activity of an organ is dependent on the integrity of its blood-vessels. The span of life is determined by the so-called vital rubber of the arterial tissue, and justifies the oft quoted axiom, "A man is only as old as his arteries." Not long ago I examined a youth of ten who was practically an old man with his rigid and incompressible radials. The pathologic process of arterio-sclerosis is essentially a chronic process leading to an increase of arterial connective tissue involving primarily the internal coat (intima) followed by calcareous infiltration. Sclerosis of the veins (phlebo-sclerosis) may be primary or secondary to the same changes in the arteries. Arterio-sclerosis is often an hereditary affection aided by factors which result in the misuse of arteries. Among the common causes are: 1. Chronic intoxications: lead poisoning, syphilis, alcoholism, uric acid, etc., which by augmenting the resistance in the peripheral vessels, raise the arterial pressure.

2. Overeating is regarded as a frequent cause, the excess of food and fluid ingested fill the blood vessels to repletion. 3. Inordinate muscular work leads to peripheral resistance with consequent rise of blood pressure. 4. Bright's disease may lead to primary or secondary arterial degeneration. The causal factors predominating in males, it is but natural that the latter are the chief victims to the affection.

SYMPTOMS.

An acute arteritis is almost never of clinical interest although some clinicians claim to make a diagnosis of acute aotitis by the fixed retro-sternal pain associated with acute disease of the aortic valves. Arterio-sclerosis is frequently a post-mortem discovery. The diagnosis rests on the general manifestations, but more often on symptoms referred to special organs, the arteries of which are particularly implicated in the sclerotic process. The accessible blood-vessels are hard and incompressible. The sensation is often that perceived in grasping a goose's neck. The pulse cannot be obliterated. The pulse may be of high tension yet no sclerosis exists. If there is any doubt, palpate the pulse with two fingers. If the artery is felt beyond the point of compression and is easily distinguished from the other tissues, its walls are sclerosed.

Next to increased arterial tension, hypertrophy

of the left ventricle is the most frequent symptom. Increased arterial tension, palpable arterial induration and hypertrophy of the left ventricle are pathognomonic of the disease. There are distinct types of arterio-sclerosis: 1, cardiac; 2, cerebral; 3, renal; and 4, peripheral types.

Cardiac sclerosis of the coronary arteries may be associated with varied myocardial lesions, notably: fibroid degeneration, angina pectoris, heart aneurism, etc. The hypertrophied heart so common in arterio-sclerosis may eventuate in dilatation followed by the usual signs of cardiac insufficiency (dyspnea, dropsy, etc.)

Cerebral. The milder symptoms are vertigo, cephalalgia, tinnitus, syncopal attacks and transient aphasia and paralysis. Thrombosis, cerebral embolism and the formation of miliary aneurisms followed by rupture are associated lesions.

Renal. The symptoms are practically those of contracted kidney, viz.: polyuria, uremic headaches and vomiting. In the peripheral type, tissue starvation leading to gangrene may ensue. Implication of the peripheral arteries in the scelrotic process does not necessarily imply that the aorta and its branches are seriously involved.

Recognition of increased arterial tension is often a matter of education. The tonometer of Gaertner is an instrument of precision in gauging blood-pressure. The blood-pressure in healthy young

persons is from 100-130 millimeters of mercury. With the tonometer, one may recognize arterio-

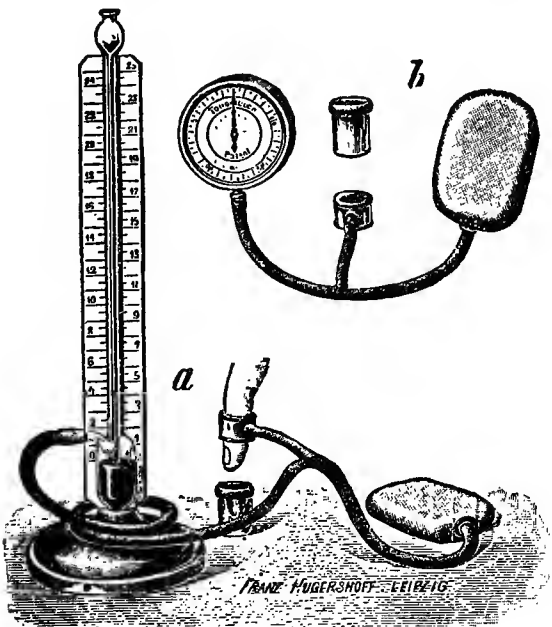


Fig. 13.

a—Tonometer provided with a mercurial gauge.
b—Same instrument more portable, provided with a metal gauge.

sclerosis without palpable changes in the peripheral vessels. When the tonometric figures are

low with clinical evidence of arterio-sclerosis, it is a sign of failing heart power.

Anyone mechanically inclined can easily construct a tonometer at small expense. The one I have used for some time, I am indebted for to Dr. A. W. Perry of San Francisco, who made several for his medical friends.

TREATMENT.

The causal factors must be considered. A history of syphilis suggests the iodides, which may generally be recommended as routine treatment. For the high pulse tension, nitro-glycerin. Venesection is indicated in instances of very high tension associated with plethoric symptoms. Use may also be made of the Schott methods.

Aneurism of the Thoracic Aorta.

ETIOLOGY AND PATHOLOGY.

The etiology is concerned with the same factors predominant in arterio-sclerosis. Alcohol, syphilis and overwork, single and in combination, furnish the impetus for arterial changes conducive to aneurism. The different varieties of aneurism are of greater interest to the pathologist than the clinician. The thoracic portion of the aorta, according to Lyman, is implicated in 75 per cent of the cases of aneurism. Within the chest nearly 60 per cent of the cases originate in the ascending

portion of the aorta, while nearly 30 per cent are seated upon the arch of the vessel.

SYMPTOMS.

Bramwell's clinical division of aneurism is a practical one: 1. Latent aneurisms which give no physical signs. 2. Those presenting signs of intrathoracic pressure but in which it is difficult or impossible to determine the nature of the lesion producing the pressure. 3. Aneurisms with marked pressure symptoms and external signs. Our primary object is to make the diagnosis of thoracic aneurism and later to define its site. The former object is attained by the recognition of pressure symptoms and objective signs.

PRESSURE SIGNS.

Pain is an important and almost constant sign. When dependent on pressure or stretching of the nerves, it is sharp and lancinating and may be paroxysmal owing to alterations in the intra-aneurismal pressure. When due to pressure against the bony structures, it is a continuous gnawing or boring pain.

Cough. Usually paroxysmal. When due to pressure on the recurrent laryngeal nerves it is of a brazen ringing character. Pressure on the trachea or bronchus may also provoke a cough.

Dyspnea owes its origin to one of the following causes: 1, Tracheal compression; 2, compression

of the left bronchus; 3, pressure on the recurrent laryngeal nerves.

Venous enlargement of the veins of the head and arm occurs when the vena cava is compressed.

Edema of the right arm occurs when the subclavian vein is compressed. Localized edema of the chest may be present.

Aphonia and dyspnea occur when the right laryngeal nerve is involved. Pressure of the left recurrent laryngeal causes paralysis of the corresponding cord with aphonia. Pressure on the sympathetic nerve causes pupillary contraction; on the thoracic duct, wasting, on the esophagus, dysphagia, on the left bronchus, bronchiectasis with bronchorrhea.

PHYSICAL SIGNS.

Inspection. With an abnormal pulsation, a tumor may be visible. The apex beat is displaced from pressure.

Palpation. In deep-seated aneurisms, pulsation is best detected by bimanual palpation, one hand over the spine and the other on the sternum, at the same time exerting pressure with the hand on the sternum. In addition to the pulsation one may feel the diastolic shock, a valuable sign.

Percussion yields the most reliable evidence. Dullness amounting to flatness can be obtained over a superficial aneurism, the area of dullness depending of course on the situation of the sac.

Auscultation. A murmur if present is systolic in time with maximum intensity over the area of dullness and, transmitted in the direction of the cervical vessels and along the course of the aorta; a coexistent diastolic murmur is usually associated with aortic insufficiency.

In the peripheral arteries the volume of the pulse is lessened. The pulse in the two radials may show differences in volume and time.

Among the recent signs are the following: 1. Tracheal tugging. The patient's head being inclined forward to relax the neck and the cricoid cartilage is grasped between the thumb and index finger, the trachea at the same time drawn upward, when, if aneurism is present, a pronounced ascending motion will be felt at each pulsation. During the maneuver, breathing must be suspended and care must be observed to avoid mistaking the transmitted pulsations in the cervical vessels. Ewart modifies this method with advantage. The observer stands behind the patient steadying the latter's head against his body and grasping the cricoid cartilage as before. In health, the symptom is only slightly present if at all. 2. Obliteration of the pulse in the abdominal aorta and its branches. When this sign is present, the aneurismal sac acts as a reservoir annihilating the ventricular systole and converting the intermittent into a continuous stream (Osler). 3. Systolic mur-

mur heard in the trachea or at the patient's mouth when opened (Drummond). 4. Tying the extremities or compressing the femorals and axillary arteries will intensify the pressure symptoms. 5. Intra-thoracic auscultation. An esophageal tube with a large aperture at the end is introduced into the esophagus and connected with a stethoscope. Aneurismal pulsation and murmur are heard (Richardson). 6. Systolic pulsations in the larynx and trachea are heard (Oliver). 7. The X-rays furnish trustworthy evidence. I have frequently detected thoracic aneurisms by their aid when no sign was present. Of course errors are as frequent by this as by other methods, but a thorough mastery of chest radioscopy is the only reliable means of eliminating mistakes.

LOCATING THE SITE OF AN ANEURISM.

Ascending aorta. Pressure symptoms evident by distension of the veins of the neck, head and arms. Displacement of the heart outward, forward and upward. Appearance of tumor and dullness to the right of the sternum in the upper second or third intercostal spaces.

Transverse portion. Intense pressure symptoms owing to the relatively shorter antero-posterior diameter of the chest at this point. The tumor may appear in the jugular fossa. Area of dullness over the manubrium or along the left sternal border.

Descending portion. Pressure signs are slight. Evidence of vertebral compression with intense pain. Dullness, if present, appears at a point on the left side of the spine at about the eighth dorsal vertebra.

Prognosis. Usually fatal. Spontaneous cure, rare. Death from pressure symptoms or rupture.

DIAGNOSIS.

From pulsation of the aorta seen in aortic regurgitation; often difficult. In such instances defer diagnosis until tumor is unmistakable. Aortic pulsations in neurotic subjects: Negative signs of aneurism. Pulsating empyema: In this affection, throbbing is diffuse, moving the entire side; pulsation not expansile; absence of murmur and diastolic shock; hypodermic needle shows pus. Solid tumors: Pressure phenomena less marked; if tumor shows pulsation it is not expansile nor attended by the auscultatory signs of aneurism; tracheal tugging is absent.

TREATMENT.

Rest and a restricted diet. A low diet such as suggested by Tufnell reduces intra-aneurismal pressure and favors coagulation. Potassium iodide (10 to 20 grains, 3 times a day) is of undoubted value. Venesection often gives relief to pressure symptoms. Insertion of wire into the sac with the use of electrolysis according to the Loreta method has given me good results in two cases.

My colleagues Kerr and Rosenstirn of this city have also reported cures. Thorne and others have reported much improvement following the use of baths on the Schott principle.

A method of recent introduction and worthy of some consideration is the use of gelatin injections. In 1895, Dastre demonstrated that if a solution of gelatin is injected into the veins of a dog, it made the blood more coagulable. The solution for injection consists of a 1 per cent sterilized solution of gelatin in a 0.1 per cent solution of sodium chloride. Of this solution, from 2-5 ounces is injected every third or fifth day, according to the reaction, into the sub-cutaneous tissues. No danger attends this treatment beyond the possibility that a clot may be carried into the general circulation. The solution for injection is placed in a flask, which is sealed and then sterilized. When ready some of the solution is introduced into a flask fitted with a cork and two tubes like a wash-bottle. To the long tube a sterilized needle is attached and to the short tube, a rubber air ball. The flask is introduced into a water bath to liquefy the gelatin and while kept there, the injection is begun.

The calcium salts have recently been recommended. Marked improvement in one of Cohen's cases followed the use of hydrated calcium chloride in doses of 1 dram daily.

CHAPTER VIII.

ADDENDUM.

The Heart Reflex.

Three years ago attention was directed to an heretofore undescribed clinical phenomenon which I called the heart reflex. It is practically a myocardial contraction consequent on irritation of the skin of the precordia by vigorous rubbing with the finger or better still by a spray of ether and is manifest by the Roentgen rays and the fluoroscope. It can be most easily provoked in children. The contraction of the myocardium is of sudden and momentary duration and, like other reflex acts, soon becomes exhausted. My assistant, Dr. Louis Gross, and myself saw both ventricles recede fully 1½ inches on either side after directing a spray of ether on the precordia in an emaciated girl of 14 years. Of course the anatomic heart in the adult measures only 3½ inches in breadth, but we are here concerned with the physiologic heart.

To properly appreciate the phenomenon of the heart reflex for therapeutic and diagnostic purposes, attention must be directed to the lung reflex.* If the skin of the thorax is irritated, arti-

*New York Medical Journal, Jan. 13, 1900.

ficial lung dilatation ensues. The degree of dilatation varies with the severity and extent of cutaneous irritation. Vigorous rubbing of the skin of the precordia is sufficient to obliterate the area of superficial cardiac dullness, if irritation is made over the lower lung border, percussion will show in the axillary line a descent of the lower lung border fully 6 cm., a degree of dislocation even exceeding that obtained by forced inspiration. Aside from its percussional recognition, the appearance of the reflex by means of the Roentgen rays is distinctive. Coincident with the discharge of the reflex, the lung area implicated shows increased brightness lasting from a few seconds to four minutes, the lung after that time assuming the normal skiascopic appearance.

In a recent contribution,* I espoused the theory that the real factor involved in balneo- and mechano-therapeutics (Schott treatment) was dependent on cutaneous irritation provoked by exercise and baths. In accordance with this theory, I have since this contribution employed vigorous cutaneous friction by means of a rough towel after immersion of the patients in a warm bath (15 minutes duration) in cases of chronic heart disease with results emulating the conventional Schott method. By my simple and expeditious method, relief of dyspnea follows, there is reduction in

*The Medical News, Jan. 7, 1899.

cardiac volume and a marked reduction in pulse rate with increase in volume and force. The accompanying illustration is a rough reproduction obtained in a young man with a massive dilatation of both ventricles; a, represents the percussional area of the heart. The dark area represents the

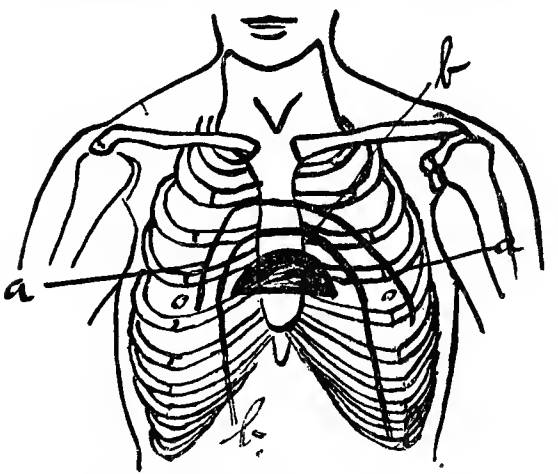


Fig. 14—Illustration of Heart Reflex.
a—Percussional area of dilated heart.
b—Area after application of cutaneous irritation.

area of cardiac dullness after directing a spray of ether on the skin of the precordia and is caused not wholly by a reduction in cardiac volume but by the lung reflex which induces the dilated lung to encroach on the area of cardiac dullness. After

waiting ten minutes, a time exceeding that necessary for the lung to recede, the percussion area, b, is obtained, which actually represents the decrease in the area of the heart. Like results follow treatment by cutaneous irritation.

This illustration will serve to exemplify the aid which this phenomenon furnishes in the differential diagnosis of a pericardial exudate from a dilatation of the heart, and I regard this heart reflex test as pathognomonic and far exceeding all other methods yet recommended in differentiation. One of the most difficult problems for the clinician is to distinguish between a dilatation of the heart and pericardial effusion. If in a given case of increased cardiac dullness which has been carefully outlined, we direct a spray of ether on the skin of the precordia and note after four minutes (the time necessary for the lung reflex to be abolished) a reduction in the area of cardiac dullness however slight, we are justified in concluding that we are dealing with cardiac dilatation and not with a pericardial effusion. The heart reflex is also a valuable index to the state of the myocardium.

Relation of Diseases of the Heart to Other Diseases.

Abdominal Typhus.—Myocarditis is often responsible for sudden circulatory collapse. Pulse rate not in proportion to temperature. Average

rate, 84-110 per minute. Pulse of more than 130 for some days is an ominous sign. Pulse dicrotism, characteristic but not pathognomonic. During convalescence a sub-normal pulse rate is frequent. Venous thrombosis occurs in one per cent of all cases (Murchison). It is the result of cardiac failure and implicates most frequently the femoral veins. Peri and less often endocarditis are complications.

ANEMIA (PERNICIOUS).—Hemic murmurs constant. Visible arterial pulsations. Pulse full and suggests the water-hammer beat of aortic regurgitation. Capillary pulse often seen. Superficial veins prominent and may pulsate.

BRIGHT'S DISEASE.—Anemia an early symptom. In the chronic forms, pulse tension increased and arterial wall thickened. Persistent high tension is one of the earliest and most important symptoms of interstitial nephritis (Osler). Hypertrophy of the left ventricle common.

CHLOROSIS.—Palpitation of the heart. Increase in the area of cardiac dullness. Systolic murmur heard in second left interspace, accompanied sometimes by a pulsation, and is produced at mitral orifice by relative insufficiency of the valves accompanying dilated ventricles (Balfour). Over right jugular vein, a continuous murmur (bruit de diable or humming-top murmur). Pulsation in peripheral veins and a tendency to thrombosis,

usually in the femoral, occasionally in the longitudinal sinus.

CHOREA.—The theory is gaining ground that chorea is a rheumatic manifestation. Pericarditis and endocarditis frequent complications, the latter occurring in about one-half of all cases (Osler). The murmurs may also be due to anemia. Osler examined 140 persons having suffered at least two years previously from chorea. In 51, heart normal; in 72, signs of organic lesion; in 17, cardiac disturbances.

DIABETES.—Plasma of blood is loaded with fat (lipemia) which form fat emboli in lung capillaries. Heart changes not characteristic and endocarditis is infrequent. Nutritional disturbances cause arterio sclerosis.

DIPHTHERIA.—Myocarditis and degeneration and endo and pericarditis may occur. Cyanosis and heart failure may be sudden. Myocarditis as a post-diphtheritic manifestation. Sudden death often caused by changes in vagus or its cardiac branches (neuritis) and may be a sequel of the mildest cases.

DYSPEPSIA.—Flatulency may cause mechanic disturbance, viz., cardiac dyspnea or pseudo-anginal attacks. The pulse will be found weak and heart tones enfeebled. The left inter-scapular sign is present (Abrams). Heart neuroses frequently owe

their genesis to the absorption of the products of indigestion.

EMPHYSEMA.—In no other affection other than in congenital heart disease is cyanosis so marked and this, with comparative comfort of the patient. Dilatation and hypertrophy of right ventricle; later, hypertrophy is general.

EXOPHTHALMIC GOITRE.—Cardio-vascular disturbances occur early. Heart sound intense and may be heard as far as four feet from patient (Graves). Throbbing of carotids and abdominal aorta. Hypertrophy of heart and murmurs at base.

GOUT.—Arterio-sclerosis, common. Blood tension persistently high leading to ventricular hypertrophy, rupture of vessels (apoplexy) and aneurism.

GRIPPE.—In the "influenza heart" cardiac weakness is very alarming and out of proportion to the height of the fever. Pulse feeble and intermittent and may persist after convalescence.

HYSTERIA.—Increased heart rapidity on slightest emotion. Pain in precardia may simulate angina. Stigmata or hemorrhages in the skin and flushes are vaso-motor phenomena.

ICTERUS.—Slow pulse (30 or even 20) common. Ecchymoses in severe forms.

INSANITY.—Mental symptoms often associated with heart disease. Delirium, hallucinations and morbid impulses (suicide) frequently terminate the

close of the disease. Insanity may develop in aortic and mitral disease in the stage of compensation.

MARASMUS.—In the terminal stages of chronic diseases, thrombosis may occur in the sinuses (marantic thrombus).

NEURASTHENIA. — Cardio-vascular symptoms often predominate. Palpitation, irregular and rapid action of the heart with cardiac pains. Slightest emotional disturbances excites heart and it is difficult to dissuade neurasthenics that there is no organic lesion. A throbbing aorta is a prominent symptom and is so pronounced as to suggest aneurism. Flushes of heat and hyperemia of skin common as vaso-motor phenomena.

PHTHISIS.—False or cardio-pulmonary murmurs result from the contraction of a lung cavity causing heart dislocation. The indurated lung intensifies the murmur. A systolic murmur at the apex may simulate mitral regurgitation. The murmur is usually caused by the impact of the heart upon partially consolidated lung tissue driving out the air. A murmur of this kind is superficial, most distinct during expiration and is inaudible when the patient lies down. In neurotic phthisical persons a false systolic murmur is heard at the apex when the heart action is excited. Chronic valvular cases, the mitral being most frequently involved (17 times in 20 cases). Congenital stenosis of pul-

monary orifice is frequently associated with this disease.

PLEURISY.—With effusion either dislocates the apex or the whole heart. There is no twisting of the heart but a dislocation of the mediastinum which carries the heart with it.

PNEUMONIA (CROUPOUS).—Average pulse rate, 100-110. Heart failure is manifested by increased frequency (120 or more). Failure of right heart-chamber is indicated by dilatation of this ventricle, viz.: increased dullness to the right, epigastric pulsation, systolic murmur, fetal heart sounds, especially second pulmonic sound and venous stasis. It is the right ventricle which needs watching and any evidence to be gained is derived by frequent auscultation of the pulmonic tones and not by palpitation of radial pulse.

RHEUMATISM.—Endocarditis is the most frequent complication, and the mitral segments are most frequently involved. Pericarditis is especially frequent in children, and is attended by a peculiar delirium. Myocarditis is commonly associated with endo-pericardial changes.

SPINAL CURVATURE.—Curvatures of the spine result in circulatory and respiratory disturbances. The heart of hunchbacks is usually increased in size and the right heart is generally dilated, resulting in disturbances in the pulmonic circulation.

SYPHILIS.—In the heart, gummata frequently involve the left ventricular wall and are usually encysted. A fibro-sclerotic myocarditis may cause sudden death. Syphilitic endocarditis is not infrequent. Arterial syphilis may occur as an obliterating endarteritis or as a gummatous periarteritis implicating the coronary, cerebral and other arteries.

THE URIC ACID DIATHESIS has, of late, assumed an important position in clinical medicine. Palpitation of the heart is frequent, particularly after eating, and increased arterial tension is an early and prominent symptom. Its occurrence during an uric acid storm is pathognomonic and this fact I have learned to appreciate since using the tonometer. Circulating uric acid produces universal arterio-spasm followed sooner or later by the well-known symptomatic complex—arterio-sclerosis, gout and contracted kidney.

CLINICAL MEMORANDA OF THE CARDIO-VASCULAR SYSTEM.

THE PULSE.

1. The number of pulse and heart beats in a normal adult is 71-72 per minute.

2. The pulse frequency in different ages in the male: 0-136; 5-88; 10-15, 78; 15-20, 69.5; 20-25, 69.7; 25-30, 71; 30-50, 70. (Quetelet.)

3. In the female, the pulse frequency is greater by from 1 to 4.5 beats a minute.

4. Influence of position on pulse: In sitting posture, 5 beats more a minute than in recumbent position; standing, 9 beats more than while sitting and 14 more beats than in recumbent posture.

5. Influence of activity: Slight activity increases beats 10-20 and running may increase the beats to 140 and this increase may last from ½-1 hour.

6. Influence of food: After dinner the average increase is 16.

7. Influence of the barometer: A barometric rise of 1½ cm. increases the pulse frequency 1.3 per minute (Vierordt).

8. In sleep the pulse is slower, especially in children.

9. In fever the pulse rises synchronously with the temperature and averages an increase of 10 beats for every degree above 98 deg. F. Expressed according to centigrade: P=80+8 (T—37).

10. Relation in time of heart tone and radial pulse beats in seconds: Radial pulse, 0.224 later than the first cardiac tone. The left is felt 0.01-0.03 of a second later than the right radial pulse.

11. Relation of respiration and pulse: 1:3½-4. It takes four times as long for the blood to go through the systemic as through the pulmonic circulation.

THE HEART.

1. The work of the right ventricle is one-eighth that of the left.

2. The intensity of the heart tones is as follows, beginning with the loudest: 1, systolic mitral; 2, systolic tricuspid; 3, second pulmonic tone; 4, second aortic; 5, second mitral; 6, second tricuspid; 7, systolic pulmonic; 8, systolic aortic tone.

2. Relative intensity of second sounds at base. Second pulmonic sound invariably accentuated in young children and frequent in youth. After the fortieth year it is rare to find a pulmonic second sound as loud as the corresponding second aortic sound. Between 20 and 30 there is no marked accentuation of either sound (Creighton).

3. In embolism from endocarditis of left heart the organs are affected in the following proportions: Kidney, 57 times; spleen, 39 times; brain, 15; skin, 14, and liver and intestines, 1 time (Sperling).

4. Location of endocarditis in 300 cases: Mitral valve, 255 times; aortic valve, 129 times; tricuspid valves, 29 times; pulmonary valve, 3 times (Sperling).

5. Endocarditis in the sexes: In 238 cases, 86 males, 152 females (Willigk); in 230 cases, 118 males, 112 females (Bamberger).

6. Influence of pregnancy on heart affections from 84 observations by Porak: Condition sta-

tionary in 25% of the cases, temporary aggravation in 4.76%, persistent aggravation, 60.71%; improvement during child-bed, 26.19%; cardiac symptoms aggravated by labor, 13.09%. Death occurred before delivery, 5 times; during delivery, 2 times; during child-bed, 25 times; after temporary improvement, 8 times. The foregoing table refers to pronounced cardiac lesions.

7. Insanity and heart disease: Among 68 cases of melancholia, 11 cases of heart disease (Esquirol); among 100 insane, 31 cases (Calmeil); 602 insane, 75 cases (Vienna asylum).

CHILDREN.

1. The movements of the heart begin one-eighth of a minute after birth.

2. The normal apex beat is usually in the fourth interspace just outside the mammary line. This position has been attributed to the greater relative narrowness of the infant's chest in the transverse diameter and the relatively larger heart. The than in the adult. Symington contends in opposition to the current belief that the position of the heart and great vessels is the same as in the adult.

3. Functional disorders: Up to the seventh year cardiac action during sleep is often of unequal strength and rhythm and prone to be irregular in the healthiest children during sleep and greatly influenced by breathing (Da Costa). Irregularity

during waking hours indicates cardiac disorder unless there are evidences of meningeal disease (Smith).

4. Bulging of precardia more frequent in cardiac diseases of children owing to flexibility of thorax.

5. Aneurisms under the age of 20, if not traumatic in origin are caused by embolism from a preexisting endocarditis. The cerebral arteries are most frequently involved.

6. The ductus arteriosus is not obilterated as a rule until two weeks after birth. Persistence of the same after the first month is pathologic.

7. Most frequent congenital lesion is stenosis of the pulmonary artery.

8. The most common evidence of a congenital heart lesion is cyanosis. It affects more male infants—180 cases, two-thirds males (Aberle). Cyanosis does not always commence at birth and may be retarded in appearance for years. Clubbing of the fingers and toes and a pigeon-chest are two common abnormalities in cyanosis. Other general conditions are lack of heat and retarded development.

9. Prognosis in cyanosis: Thirty-five per cent die before the end of the first year; more than two-thirds die before the age of eleven years, and only five lived more than 45 years (in 159 cases collected by Aberle). The most frequent modes of

death are convulsions, dyspnea, hemorrhage, coma and phthisis.

10. Most frequent lesions in cyanosis: Stenosis of pulmonary artery, transposed aorta and pulmonary artery, one auricle and one ventricle, right ventricle divided into two cavities by a supernumerary septum. In more than half the cases the lesion is located in the pulmonary artery.

STETHOPHONOMETRY.

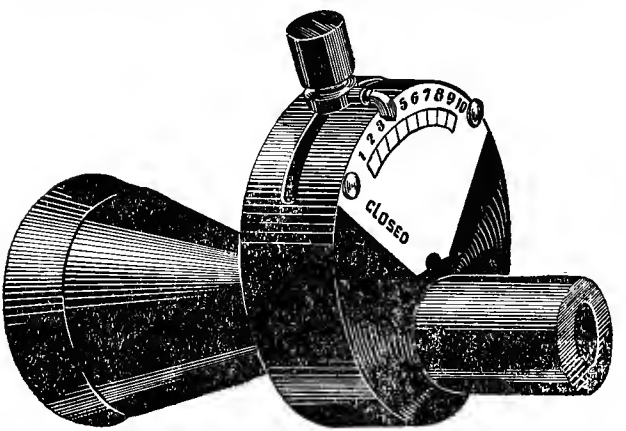
Since reference was made, on page 55, to methods of measuring the intensity of the heart tones, I have had constructed for me, after considerable experimentation, a simple stethophonometer, which can be readily attached to any stethoscope.*

STETHOPHONOMETER.

The stethophonometric attachment weighs about two ounces and is based on the principle of a disc valve with attachment on one side for the stethoscope and on the other side for the bell. The

* Made by the Shoenberg Electrical Co., E. Spreckels Building, San Francisco.

object of the valve is to offer resistance to the sound waves. There are three hard rubber discs, all of which are perforated. The center disc is easily movable by means of a handle so as to carry the opening away from the other two discs. On the face of one of the discs is a graduated scale, which enables one to measure the intensity of the



STETHOPHONOMETRIC ATTACHMENT.

cardiac tones. If the heart sounds are loud enough to overcome the resistance of the valves, further resistance may be offered by the insertion of a small rubber cork in the bore of the plug which fits into the stethoscope. It will rarely be found necessary to make use of the latter expedient.

INDEX.